The Journal

of the

Louisiana State Medical Society

$4.00 Per Annum, 35c Per Copy
Vol. 105, No. 2

FEBRUARY, 1953

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

SPINAL HEADACHE*

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Headache following anesthesia, while usually not a serious complication, certainly is annoying and disturbing to both patient and anesthesiologist. Headaches do occur following general anesthesia as well as spinal anesthesia. The headache folowing spinal anesthesia, or even simple lumbar puncture, usually is bitemporal, bifrontal or occipital, either brought on, aggravated, or relieved by changes in position on the part of the patient.

Interest in this complication seems to run in decade cycles. In 1902, Secard[17] reported that postpuncture headache was due to cerebral spinal fluid leakage at the puncture site. In 1923, Antoni[1] suggested the use of smaller needles to decrease the incidence and degree of leakage. In 1926, H. Green[9] reported on the histology of the dural fibers and the influence of puncture trauma on headache. Within the past five years, particularly since the work of the Swedish author Thorsen,[19] there have appeared in the medical literature articles by Graf,[7] Hebert,[12] Haraldson,[11] B. Greene,[8] Whitacre,[21] Nicholson,[14] Shaw,[18] and many others, as to the etiology, prevention and treatment of this complication, postspinal or postlumbar puncture headache.

The Swedish authors, Thorsen[19] and Haraldson,[11] call attention to two distinct types of headaches. The first type and less frequent, 20 per cent of their cases, is headache caused by a marked increase in cerebral spinal fluid pressure. The second and more common, 80 per cent in their series, is the result of a marked decrease in cerebral spinal fluid pressure. These authors describe the headache due to marked increase in cerebral spinal fluid pressure as splitting in nature, readily influenced by the ordinary analgesics and definitely not influenced by positional changes on the part of the patient. Again, the second and more common type of headache, that associated with a marked decrease in spinal fluid pressure, is described as dragging and dull in nature, markedly influenced by positional changes; that is, aggravated by rising, diminished or relieved by the head-down position and responding very poorly to the ordinary analgesics. This second type, the type of extreme low pressure, is universally recognized and accepted by all authors. The first type, the type of increased pressure, is debatable and questioned by some authors.

Both types may best be reviewed as to etiology under five general headings or theories:

1. Purely mechanical.
II. Chemical, that is chemotoxic.
III. Inflammatory (traumatic).
IV. Psychic suggestion
V. Systemically disturbed fluid balance.

In all five theories, we have overlapping and occasionally do have contradictions.

I. MECHANICAL THEORY

In 1929, Evans postulated the theory of spinal fluid leak on a purely mechanical basis. It is known that the dural and subarachnoid tissues have very little elastic tissue component. In the positioning of a patient for lumbar puncture, there is a slid-

*Presented at the Fifteenth Annual Meeting of the New Orleans Graduate Medical Assembly, March 1952.

ing effect of dura over arachnoid tissues. Return to normal positioning reverses this sliding effect with the resultant sealing of the double puncture hole. In patients with low pressures or relaxed tissues, this sliding effect does not take place, and a fistula between the dura and arachnoid tissues persists, with leakage of fluid into the epidural space. Most authors feel that this is but a part of the spinal headache picture.

II. CHEMICAL THEORY

Some authors believe that there may be a direct chemical effect not only on the nerves but also on the vascular structures responsible for fluid secretion, through the choroid plexus in the ventricles; or fluid excretion by way of the arachnoid villi or pacchionian bodies. That the agent may influence directly as high as the choroid plexus is postulated from Novak's experiment with animals. He completely transected the spinal cord and enveloping tissues so that there was no direct communication between the severed sections. He then injected an anesthetic drug into the subarachnoid space of the distal segment and recovered a qualitative specimen of the drug in the spinal fluid within the ventricles. Foldes[6] is about to publish work on controlled decreases in cerebral spinal fluid pressure. He feels the causes of spinal headache are more than the local mechanical leakage of spinal fluid plus dehydration. His findings suggest important factors of chemical equilibrium within the subarachnoid space; that is a disturbance in cerebral spinal fluid secretions; or absorption of the cerebral spinal fluid.

This theory of chemotoxic effects would conveniently explain the instances of both high and low pressure types of headache. The action of the anesthetic agent might result in choroid plexus hyperemia and an increase in its secretory activity, in patients with high pressure headaches, or the opposite may occur; hyperemia and secondary edema with obstruction may occur, resulting in a decrease in secretory activity; this combined with fluid leakage at the puncture site would cause the low pressure type of headache. Thus, we have here contradiction to explain one's preference of theory.

III. INFLAMMATORY OR TRAUMATIC THEORY

Most authors accept this theory as one cause for low pressure headache. Haraldson[11] claims that blood, trauma or inflammation resulting from repeated puncture may cause a definite increase in spinal fluid pressure due to inflammatory reactions within the subarachnoid space. Work by Arrowood and Foldes[2] in their differential spinal investigation demonstrated that pressure increases over 300 mm. of mercury do not result in headache. As to the influence of trauma and blood, an amusing and possibly pertinent observation by one of our "elder" neurologists in Boston states that the absence of headache occasionally noted following a traumatic puncture may be due to the sealing off effect of the blood at the point of puncture. As opposed to this postulation, Hebert[12] shows a definite per cent of increase in postspinal puncture headache following multiple traumatic punctures to the patient.

IV. PSYCHIC SUGGESTION

Fear of headache, past experiences or unconfirmed reports of friends or relatives may play an important part in the incidence of postspinal headache. Interesting enough, the higher the I.Q. of the particular patient the more frequent the headache. The emotional tension present in our mode of living plus subconscious fear is an influencing factor. Again, the frequent ill-advised warnings relative to headache also play an important part in suggestion.

Add to psychic suggestion, the physical discomfort of the extreme head-down position or prolonged hyperextension of the patient's neck, frequently ordered by many doctors, and we have a patient developing a severe headache. This neck strain may act as a continuous reminder of headache possibilities. Certainly, a small pillow is more comfortable to most patients than exaggerated hyperextension of the neck. The third type of suggestion is the patient association, as reported by Allan Conway[4] in discussing Hebert's[12] paper in Atlantic City in 1949. To quote: "We found that when one patient had a headache in a big ward several more patients would have it in the next couple of weeks." Conway cited his

army experience of a chronic complainer with headache upsetting his adjacent bedmates. These bedmates previously had spinal anesthesia without headache and subsequently had five and seven spinal anesthesias respectively without headache.

V. SYSTEMIC FLUID IMBALANCE

This theory is one of disturbed fluid balance due to excessive leakage at the puncture site and a systemic decreased hydration associated with electrolyte imbalance. The factor of local leakage had been long recognized by Secard[17] in 1902, Antoni[1] in 1923, H. Greene,[9] in 1926, and others. Greene demonstrated that the majority of the dural fibers run longitudinally. A puncture at right angle to these fibers, that is, a cross cut, by the average needle results in a greater rent or tear, and the delayed healing of puncture hole. Thorsen,[19] in 1947, definitely demonstrated this leakage with the injection of lampblack into thesubarachnoid space and recovering the colored fluid in epidural pockets, both at postmortem examinations and in patients undergoing laminectomies.

The occurence of systemic decreased hydration has been recognized by the neurosurgeons since the report of Weed,[20] in 1919, and Cushing,[5] in 1920. They reported that cerebral spinal fluid pressures can be increased by hypotonic solutions given either intravenously or by mouth and that the pressure and volume can be decreased by use of hypertonic solutions. Wallace Shaw[18] in 1951, stated that decreased fluid intake plus electrolyte imbalance, particularly salt loss are important factors in spinal headache. The question of postoperative salt requirements at the present time is debatable. Moore states that salt is not needed for the first forty-eight hours unless there is excessive gastric drainage with chloride loss.

Most authors now feel that the combination of local fluid leakage plus systemic decreased hydration result in a state of cerebrospinal fluid hypotension. This hypotension results in: (1) secondary venous engorgement within the cranium with stretching of the vessels; (2) loss of the cushioning effect of cerebrospinal fluid on meninges and nerve roots; (3) a stretching of nerves within the cranium. All three conditions individually or combined may result in headache.

While the theories of causation are of interest to the anesthesiologist; to the patient the best therapy is prevention. All authors quote percentage ranges of from 6 to 20 per cent decrease in the occurence of headache following their recommendations for prevention. All recommendations can be summarized as follows: In the hypertonic patient or the patient with increased pressure, avoid trauma or inflammation. In the more commonly accepted type, that is, 80 or 90 per cent of the patients, with reduced or very low pressure, lessen leakage and increase systemic hydration.

Recommendations of many authors to lessen this local leakage of spinal fluid can best be adhered to by observing the following three precautions. The rent in the dura should be as small as possible. The subject should remain completely immobile during the puncture attempt so as not to enlarge the hole. There should be only one hole in the dura. H. Greene,[9] in 1926, recommended bevel penetration of the dura parallel to the longitudinal fibers. He also modified the routine spinal needles to a 90 degree angle with a sharp point and rounded lip of the bevel; in order to nick with the tip of the needle and spread these fibers with the remainder of the blunted bevel. Since Antoni's[1] recommendation in 1923, the trend has gradually been toward smaller and smaller gauge needles. At the present time B. Greene,[8] in 1949, reports on the use of 24 and 26 gauge needles with marked reduction in the incidence of spinal headache from 6 per cent to 0.4 per cent. One drawback to the use of a needle with such a fine caliber is, due to the extreme slow fluid flow, occasionally one cannot be certain the tip of the needle is within the subarachnoid space. This may result in a higher incidence of a poor to no spinal anesthesia. Another danger with the use of an extremely fine needle is needle trauma or needle breakage Needles of this extreme fine caliber require an ex-

tradural guide for better control or protection. Another drawback is that there is always the danger of introduction of needle through and beyond the subarachnoid space into the vascular plexuses located along the anterior wall of the spinal canal.

Whitacre,[21] in 1950 and 1951, reported the use of a 20 gauge pencil point solid tip needle with the hole on the side of the needle 2 mm. proximal to the solid tip. Haraldson[11] in Sweden independently reported a similar needle recommended by Professor Sjovall. Both authors suggest this type of needle separates the longitudinal fibers without trauma. This is but deductive reasoning and is not supported, as yet, by direct clinical evidence. Whitacre[21] reports the incidence of spinal headaches dropped from 5 to 2 per cent with the use of this particular type of needle. But, again, this particular type of needle may introduce technical difficulties as the hole is 2 mm. from the tip; and in the presence of preexisting low pressure, the dura may be pushed forward without the needle opening penetrating within the subarachnoid space. That is, there are dangers of either (1) an epidural injection, or (2) hemorrhage from trauma to the venous plexus along the anterior wall of the spinal canal. The presence of low pressure is not uncommon especially where there has been recent lumbar puncture, following a myelogram or a recent operation, and in patients suffering from severe systemic debilitation, dehydration, or fever.

Arrowood[13] recently presented a preliminary report on another method for prevention of local leakage. It is the injection of a variable quantity of saline, 10 to 20 cc. into the epidural space at the time of puncture, immediately following or during the removal of the spinal needle. Theoretically, this addition of epidural fluid anticipates a form of therapy employed to reduce the capacity of the subarachnoid space by increasing the epidural space pressure, thus lessening gravity factors for fluid displacement. Arrowood[13] states that this external epidural pressure tends to act in a sealing capacity on the dural puncture rent. The hazards of such a procedure or technique may be observed in the average obstetrical patient; or the patient straining or coughing. In both instances, the increased epidural venous engorgement frequently results in poor control of the height of anesthesia. Any increase in epidural pressure before fixation of the agent may result in upward progression, with medullary anesthesia, especially in the patients in whom no leakage may occur. It is fair to presume that this procedure would be ill-advised in patients with increased pressure headaches; although Arrowood[2] states, from controlled experiments, headaches do not result from increased pressure. I, personally, have had one patient with a severe pressure headache; manometric readings demonstrated a pressure of over 300 mm. of mercury. The lowering of this pressure, by the removal of spinal fluid, relieved the headache.

SYSTEMIC HYDRATION

While the prevention of local leakage is a big factor, many authors feel an increase in systemic hydration, especially in the debilitated, dehydrated or febrile patient, is necessary to prevent the onset of spinal headache. We,[10] in 1944, recommended a daily minimum of 3000 cc. fluid, plus pituitrin, for the relief of headache following subarachnoid ammonium sulphate therapy. Graf[7] advised enforced hydration by increased water intake combined with pituitrin, the antidiuretic principle of the posterior pituitary extract. This regime is used during the first three postoperative days. Wallace Shaw[18] recommended the routine administration of fluid plus electrolyte, particularly saline. He recommends, postoperatively or postpuncture, 1000 to 1500 cc. of normal saline solution, to be followed by 1000 cc. of 5 per cent glucose solution in distilled water; the rate of flow for these solutions to be approximately 500 cc. per hour. In the aged and cardiac patients, this quantity is reduced. We personally feel a minimum of 3000 cc. of fluid, that is, 5 per cent glucose in distilled water, per day is an important factor in lessening headache. I do not recall seing a spinal

headache in a Joslin diabetic postoperative patient. These patients routinely have 2000 to 3000 cc. of intravenous fluid daily.

To sum up measures of prevention:

1. No trauma on the tap.
2. Reduce the rent or tear in the dura by either using a smaller needle or special needle, or the introduction of needles in the proper parallel position.
3. Minimize psychic factors.
4. Adequate to forced hydration.
5. Promote systemic fluid retention.

TREATMENT OF THE SPINAL HEADACHE

If headaches do occur, precautions or not, what is the therapy for treatment? Therapy may be divided into the following:

1. Symptomatic.
2. Local or specific therapy.
3. Systemic or the combination of all three.

1. *Symptomatic Therapy*: The primary purpose of this therapy is the alleviation of headache usually by medicine or positional changes. The medicines consisted primarily of analgesics or narcotics; positional changes being such as low head rest to reduce headache or until the symptoms disappear.

2. *Local or Specific Therapy*: This therapy is usually confined to attempts at (a) preventing leakage or (b) increasing the subarachnoid pressure either by the intrathecal or the extradural injection of fluids. Intrathecal injection is the direct introduction of replacement fluids, usually 40 to 80 cc., within the subarachnoid space. This particular approach is recommended by very few authors as it is usually a temporary relief; and again may result in another puncture hole thus further leakage. The principle of extradural pressure with the resultant increasing of subarachnoid pressure is based on the fact that by increasing epidural pressure we compress the subarachnoid space thus raising the pressure. Many authors recommend the direct approach of the introduction of from 40 to 80 cc. of normal saline directly into the epidural space by means of an epidural lumbar puncture. Some recommend a continuous epidural infusion through a lumbar interspace. Another direct method, that of Rice,[16] is by the caudal infiltration of the epidural space intermittently by use of a plastic continuous catheter in the caudal canal. Twenty to 30 cc. of normal saline are injected as often as deemed necessary to relieve the headache. Rice[16] reports from one to three such injections usually suffices.

The various indirect methods of increasing epidural pressure are by means of increasing intra-abdominal pressure. Usually, the increase of intra-abdominal pressure causes venous engorgement of the blood vessels within the epidural space. This venous engorgement results in a secondary compression of the subarachnoid space thus raising the pressure within. Among the mechanical methods employed, to produce increased intra-abdominal pressure, are tight binders, special abdominal belts as used in obstetrics, or the simplest of all in the female, the wearing of a tight girdle.

While direct and indirect methods of local therapy are recommended, some anesthesiologists feel that the increase in epidural pressure may be transient in nature resulting in a stabilization between the fluid within and without the subarachnoid space within a period of twenty to thirty minutes.

3. *General or Systemic*: This third type of treatment is varied and occasionally contradictory. The methods of therapy may be divided thus: (1) fluids plus drugs influencing the vascular system, or (2) drug therapy alone. I have already mentioned enforced hydration for the prevention of headache; the same holds true for the treatment of spinal headache. We[10] have routinely employed the twice daily administration of 1500 cc. of 5 per cent glucose in distilled water, and when this individual quantity is down to the last 500 cc., 10 units of pituitrin are given intramuscularly. The rationale of this therapy is that the vessels of the brain and allied structures have a weaker, if any vasoconstricting action, than the vascular beds throughout the remainder of the body. The pituitrin influence on the more active vasoconstricting beds will drive fluid into the subarachnoid space. Again,

pituitrin has an antidiuretic action and thus favors fluid retention. Shaw[18] states that the added salt increases fluid retention; therefore, a greater reservoir for the choroid plexus to draw upon. Graf[7] uses 500 to 1000 cc. of 5 per cent dextrose in a hypotonic saline solution, .45 per cent normal saline, which contains 100 mg. of nicotinic acid. This combination, acting as a vasodilator, increases the blood flow through the choroid plexus.

In instances where drug therapy alone is employed, independent of fluid and exclusive of analgesics, nicotinic acid is given orally in the form of niacin 50 mg. tablets, 2 to 4 tablets, or until flushing results, and this particular dosage is repeated at four to six hours intervals until the relief of headache. My experience has been little or no relief with niacin in the severe cases of headache. Another agent employed is caffeine sodium benzoate. A few of my colleagues are trying high concentrations of oxygen since Comroe and Dripps[3] stated the possible value of high concentrations of oxygen for the treatment of migraine headaches. The high concentrations supposedly have a vasoconstricting action on the vessels of the brain. This in turn may reflexly lessen venous engorgement.

Finally, there are many methods of suggestion therapy; some highly individualistic, not the least of which is hypnosis or suggestion. Far fetched, but it will work if one is proficient in hypnosis, as was demonstrated by Raginsky[15] in Boston.

CONCLUSION

To sum up this presentation: The etiological causes for spinal headache are local fluid loss at the site of puncture plus a state of systemic dehydration varying in duration. The prevention and therapy of spinal headache is primarily the correction of the etiological causes. Lessen local fluid leakage, increase or promote enforced hydration. This will result in the secretion of cerebral fluid being greater than fluid loss.

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EN BLOC SURGERY FOR INTRA-ORAL CANCER*

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SHREVEPORT

Many therapist and cancer clinics report about 25 per cent five year cure rates for intra-oral cancer. Although such results surpass the cure rate in lung cancer and are far superior to those in gastric cancer, the outlook for three out of four individuals affected with cancer of the tongue, floor of the mouth, alveolar ridge, tonsil and tonilar pillars is dismal. Unrelenting pain, salivation, progressive nutritional failure, and foul sloughs plague the patient and

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, April 30, 1952, Shreveport.

harass his family and physician. Death results from local and regional disease and almost never from blood stream invasion. Familiarity with this picture leaves the medical profession and laity with a feeling of uncertainty and promotes delay and inadequate and improper treatment. Further discussion of some phases of the problem, along with an attempt to standardize treatment seems indicated.

REASONS FOR POOR RESULTS

To survey past experience and properly evaluate treatment, let us consider the most apparent reasons for poor results in these lesions that are so accessible to early detection by the examining eye and finger. One of the most important of these is the anatomical factor which interferes with the proper application and use of x-ray, radium, radon seed, and surgery. The proximity of the neoplasm to the teeth, bone, and functionally important organs such as the salivary apparatus and tongue, presents mechanical interference as well as sequelae.

A second and very important factor is that of infection. Infection rapidly propagates extension and metastases so that lesions readily classified as early can within four to six weeks seem almost hopelessly advanced.

A third factor of great importance is the resistance of the lesion to irradiation. Whether or not growth will continue almost unabated while intensive therapy is being applied cannot always be predicted prior to trial of therapy. A corollary to resistance of the primary is the fourth factor, that is, the ease and speed with which metastases occur. Many authors have pointed out the frequency of nodes developing and becoming fixed while the primary is being actively irradiated.

A fifth significant reason for failure to cure is less directly applicable but still significant and brings into account the difficulties of application of intensive irradiation or extensive surgery because of age and nutrition.

TREATMENT

Prior to the development of x-ray the treatment was surgical. Local removal gave poor results and it was hoped that more cures would be obtained by irradiation. Little improvement resulted and various combinations of irradiation and surgery were then proposed. Many clinics have adopted and are extending the plan of irradiating the primary and dissecting the neck. This plan has considerable merit and takes into account the high incidence of cervical metastasis. Ward and Robben report Morrow as finding tongue cancer to be present in nonpalpable nodes in 39 per cent, and in palpable nodes in 52 per cent, while oral cancer generally has 34 per cent metastasis when nodes are not palpable and 55 per cent when nodes are palpable.

Such a combination of treatments has improved results to 30 per cent five year cures in some clinics, but has failed in the remaining cases because the primary is not controlled or is not brought under control until hard and fixed metastases have developed. Also, this combination does not accomplish en bloc therapy as in radical breast surgery or in the Miles' abdominoperineal resection. Many cancer cells are in the lymphatics between the primary and the cervical region and are especially prone to become fixed to the periosteum of the mandible or to invade the bone. Robben reports that lymphatics pass from the floor to the mouth through the mandibular periosteum in half the anatomical studies conducted by Polya and Novratil.

A logical approach to fulfill the requirements of cure has resulted in the adoption of the composite operation. This procedure is designed to excise the primary, the floor of the mouth, mandible and neck nodes in continuity. It can be used in combination with irradiation to the primary or as the initial treatment. It is major, mutilating surgery but has been made feasible by improvements in surgical technique and reconstruction along with improvements in anesthesia, antibiotics, and nutritional support of the patient.

We have carried out the procedure in 8 cases. Six of these cases were operated on from the Head and Neck Department of Shreveport Charity Hospital Tumor Clinic

where the resident staff did much of the work. To a great extent the technique is patterned after that of Slaughter, Booser and Smejkai and from Ward, Hendrick and Robben. In our last 2 cases split graft linings were prepared according to Edgerton's description. The following technique reviews many of the thoughts expressed in publications by these authors.

Preliminary therapy includes evaluation of the nutritional and cardiorenal status and attention to oral hygiene. We now feel that preliminary plans should be made concerning closure of the wound and possible future reconstruction of the mandible. It is important not to unduly delay surgery but it is wise to consider ways and means of avoiding fistulae, contractures, displacements of mandibular fragments, failing nutrition and morale. This preliminary planning concerns itself with full thickness flaps prepared in the cervical skin for possible future bone graft or with split graft lining for cervical and cheek flaps when no future bone grafting is expected.

Additional planning may be directed at fixing the mandibular fragments by wire, internal plate, wiring of teeth, or by external fixation with a band around the neck attached to angles of the mandible or by the use of external splints. In most cases it will be found that no fixation is desired.

Such procedures may require ten to fourteen days and can be carried out under local anesthesia while improvement in oral hygiene and general support is being accomplished. The actual operation then is done with endotracheal anesthesia and is started by completing the neck dissection up to the hyoid level. The skin flaps are closed and the mandible removed as a part of the specimen or subperiosteally by elevating the periosteum and removing the bone proper. The dissection is then carried upward entering the oral cavity by incision above the mandible or mandibular periosteum. Where the tongue is involved it is pulled into the wound by traction suture and incised down its center. If there is extensive growth anteriorly, the anterior belly of the digastric, geniohyoid, and mylohyoid are removed. If posterior growth is greatest, the mylonyoid, styloglossus, posterior belly of the digastric and stylohyoid are removed, with the specimen. This procedure is followed when the lesion involves the anterior pillar or pharynx.

Following this resection the neck dissection is completed and internal fixation for mandibular fragments put in place if this is planned. The previously prepared flap is sutured by chromic suture to the tongue or floor of the mouth. Skin closure is secured, drains put in place and a nasogastric tube inserted. A tracheostomy is done at this stage or may have been instituted at the beginning of surgery. If the lesion is in the tongue and is small enough to be 1 cm. or more away from the nearest segment of mandible it is possible to avoid mandibular resection by a "pull through" procedure as outlined by Ward and Robben.

Seven of our 8 cases are alive and free of disease six to thirty-six months following surgery. The 1 patient who died was not free of disease at the completion of surgery.

CONCLUSIONS

The composite operation fulfills the requirements of good cancer surgery and can be accomplished without undue risk to the patient.

Our experience and the reported experience of others would seem to indicate that many cases otherwise lost can be salvaged. Generally speaking, these patients are grateful and well adjusted. They are able to carry on their usual routines.

Palliation alone is adequate reason for surgery. Should all cases be lost during subsequent follow-up, they will have escaped much of the pain, fistulae and foul necrosis characteristic of intra-oral cancer.

Review of the many problems of intra-oral cancer points out the difficulty of separating the case who will be well without en bloc surgery from the next three who will need it for cure. This would suggest the necessity of treating all cases with en bloc surgery either primarily or as an early follow-up to irradiation.

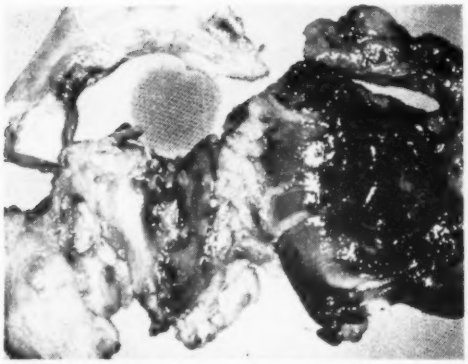

Figure 1.—Resected specimen showing mandibular segment, one-half tongue, and neck dissection. Lesion is seen at base of tongue.

Figure 2.—Postoperative. (Case No. 1, B.M.P.)

Figure 3.—Case No. 1, B.M.P.

CASE REPORTS

Case No. 1. B. M. P., 75 year old white male. This patient was first seen in Tumor Clinic on May 25, 1950. He stated that in 1948, he noticed a lesion on his right buccal mucosa. A local physician treated him with injection and referred him to a radiologist who proved epidermoid carcinoma Grade 2, by biopsy. He was then treated with 3000 R to two external ports and 1680 mgs. hours of interstitial radium. Since that time swelling in front of and posterior to the mandible progressively increased in size.

Figure 4.—Postoperative. Case No. 1, B.M.P.

Examination revealed ulceration of the buccal mucosa extending down to the alveolar process of the mandible, with exposed necrotic bone. Biopsy of the ulcerated area was negative. The mass was aspirated and reported as positive for malignant cells.

The patient was prepared for radical surgery, and on June 28, 1950, resection of right mandible and neck dissection were done in continuity. Pathology report of surgical specimen showed that the primary site of carcinoma was thought to be in the area of the healed ulcer on the alveolar ridge with metastases to a lymph node adjacent to the submaxillary gland. Sections revealed no lymph nodes. Nothing significant in the mandible.

Subsequent follow-up at regular intervals revealed no evidence of local or general recurrence and the patient has no complaints.

Case No. 2. F. W., 65 year old white male. This patient was first seen in Tumor Clinic on September 6, 1939, with a chief complaint of an ulcer on his lower lip of eight months' duration. Biopsy proved this to be squamous carcinoma Grade 2. He received 3734 R in air to the lower lip with good healing. He was followed at regular intervals for about one year and then lost to follow-up until April 10, 1951, when he was seen in Tumor Clinic complaining of a painful throat of four weeks' duration and intermittent bleeding from the region of his left tonsil of the same duration. Examination at this time revealed the left tonsil to be enlarged and slightly hyperemic, with a 0.5 cm. cavity in the anterior superior quadrant. The lesion was firm to palpation and there was a 1.5 cm. node palpable in the left cervical chain. Biopsy of the lesion of the tonsil proved it to be squamous carcinoma Grade 2 plus.

He was given intensive x-ray therapy (3000 R in air, to each of 2 ports) during April and May of 1951. He was discharged May 11, 1951, and returned in one month with good regression.

He was again admitted in June 1951, and had radon seeds implanted into the left tonsil and pos-

terior portion of the tongue. These were calculated to deliver a dosage of 5000 gamma R's. He was then discharged and seen again in July. At that time he was found to have 1 cm. indurated, tender area in the mid-lateral portion of his tongue on the left. One biopsy in this region was negative, but the second biopsy proved to be squamous carcinoma Grade 2 plus, infiltrating striate muscle.

He was then prepared for radical surgery and on September 11, 1951, he had a neck dissection with removal of the mandible, one half of the tongue, tonsil and a portion of the oropharynx, done in continuity on the left. The postoperative course was complicated by the fact that there was breakdown of the posterior portion of the wound in the superior portion. He is in the hospital undergoing plastic repair of the defect at this time.

Pathology report on the surgical specimen was squamous carcinoma Grade 2 infiltrating striate muscle of the tongue. The lymph nodes were negative for metastases.

Case No. 3. B. L., 50 year old colored male. Patient noted small swelling to right of mental process of mandible in January 1950. Two months later noted ulcer over right side of tongue. Examination revealed a huge mass, hard and nodular, in the right suprahyoid region attached to the mandible. Examination of the mouth revealed a large ulcer beginning at the base of the tongue and extending forward for 4 cms. It was 2 cms. in diameter. Biopsy was reported as squamous carcinoma Grade 2 to 3.

He was prepared for radical surgery and had a Commando procedure (one-half tongue, mandible, and neck dissection in continuity) done on April 24, 1950. Pathology report on surgical specimen showed squamous carcinoma Grade 2 to 3 of tongue with only one node positive but the submaxillary gland was replaced by tumor. Tumor tissue was left on wall of larynx. Surgery was followed by 1000 R units in air and 1300 mg. hours of radium by means of three radium needles into the tongue. This was completed on June 20, 1950.

The patient was readmitted on July 6, 1950, for excision of nodule 3 inches above clavicle on the anterior border of the trapezius, which was reported as squamous carcinoma Grade 2. Since there was active carcinoma over the carotid vessels, it was suggested that the artery be obliterated for a matter of hours to see if he could tolerate resection of the common carotid artery. This procedure was carried out and the patient tolerated the clamp well, but resection of the vessel was decided against since it was felt that tumor tissue extended beyond the limits of beneficial surgery.

Another 400 R of x-irradiation given for pallation on July 26, 1950, to a tumor nodule below the right clavicle. He was followed at regular intervals and kept well sedated with morphine sulphate. The tumor continued to grow locally and the patied expired at home of local disease on November 9, 1950.

Figure 5.—Case No. 4, R.L.B.

Figure 6.—Case No. 4, R.L.B.

Case No. 4. R. L. B., 47 year old white male. This patient was first seen in Tumor Clinic, on May 25, 1950, with a history of hospitalization two years previously for incision and drainage of a pretonsillar abscess, with uneventful postoperative recovery. For two to three months prior to admission to Tumor Clinic he had a painful ulceration in the right side of his mouth at the base of the tongue and the base of the tonsillar pillar extending over into the alveolar process. One lymph node was palpable at the tip of the hyoid bone on the right side. Biopsy of this lesion revealed squamous carcinoma Grade 3. Tumor conference recommended radical surgery.

Patient was prepared for surgery and a resection of right jaw, tonsillar fossa, and unilateral neck dissection was done on June 8, 1950, and patient was discharged on June 24, 1950. Pathological report of surgical specimen was squamous carcinoma Grade 2 to 3, arising from mucosa of buccolingual fold at anterior margin of right tonsil. Invasion was superficial extending into the submucosa and mucous glands. The tonsil was not involved. No metastases were found in lymph node.

He was followed at regular intervals after surgery, and on January 10, 1951, a small lymph node was removed from the neck just to the left of the midline 4 cm. below the mandible. This was reported as nonspecific lymphadenitis. Subsequent

follow-up to the present time has shown no local or regional recurrence but he continues to complain of inability to gain weight and some difficulty in swallowing.

Case No. 5. J. B., 70 year old white male. This patient was admitted July 3, 1951, complaining of a sore jaw. He had first noticed a mass in his left lower jaw three weeks previously which rapidly increased in size and began to drain pus into his oral cavity. He had had carious teeth for years.

X-rays revealed a large area of bone destruction in the left mandible underneath the lesion. Biopsy showed squamous cell carcinoma Grade 2 to 3.

In preparation for surgery, the teeth were extracted and an attempt made to improve his oral hygiene. He also received blood and penicillin.

On July 31, he was taken to surgery and the primary lesion, most of the left mandible, and the cervical lymph nodes removed in continuity. A primary closure was done. Pathology reported squamous carcinoma with extension to the mandible and metastases to three lymph nodes.

His postoperative course was uneventful, and he was discharged on the fifteenth postoperative day with good healing.

Follow-up has revealed no evidence of recurrence.

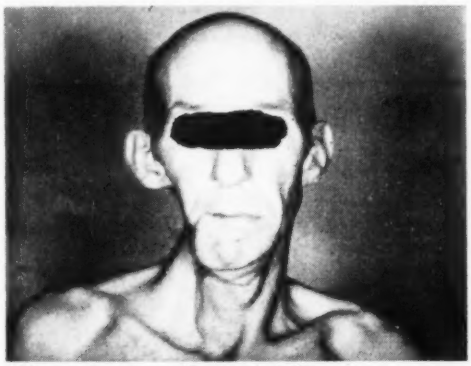

Figure 7.—Case No. 6, A.A.R.

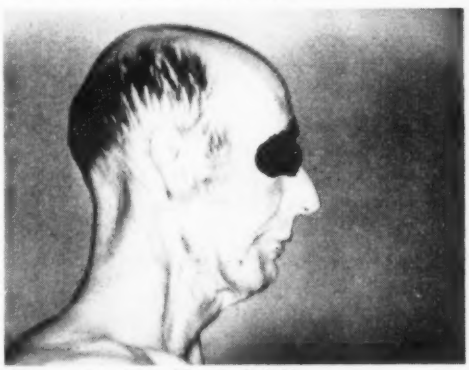

Figure 8.—Case No. 6, A.A.R.

Case No. 6. A. A. R., 43 year old white male. Patient was first seen in Tumor Clinic in May 1947, with a chief complaint of a sore in his mouth. A biopsy by a local physician was reported as squamous carcinoma Grade 4. Examination revealed a fungating lesion arising from the left tonsil and involving the base of the tongue. Palpable nodes were present in both cervical chains, the largest one being in the right midcervical region. Aspiration biopsy of the nodes on the right was positive for epidermoid carcinoma. The patient was marked for x-ray therapy to two external ports to be followed later by intra-oral treatment.

He was treated with intensive x-ray treatment to external (3000R) and intra-oral (1800 R) ports. He developed necrosis and some sloughing in the tonsillar area, but biopsies were negative for carcinoma. In May 1948, a large, firm node was noticed in the anterior cervical chain on the left. This completely regressed with x-ray treatment 4200 R. He was negative for recurrence until January 1949, when a 2.5 cm. lesion was noted on the left side of tongue 1 inch anterior to the tonsil. Biopsy showed a Grade 1 plus squamous carcinoma. It was then decided to treat the patient with surgery. On March 31, 1949, he had a resection of the left angle of the mandible, left side of tongue, and a radical neck dissection. The surgical specimen was reported by pathology as epidermoid carcinoma Grade 2 plus of the tongue with one node positive for carcinoma located in the anterior superior cervical region. Postoperatively he healed well, and repeated clinic visits failed to reveal any local or regional recurrences. He complains of discomfort in the region of the temporomandibular joint.

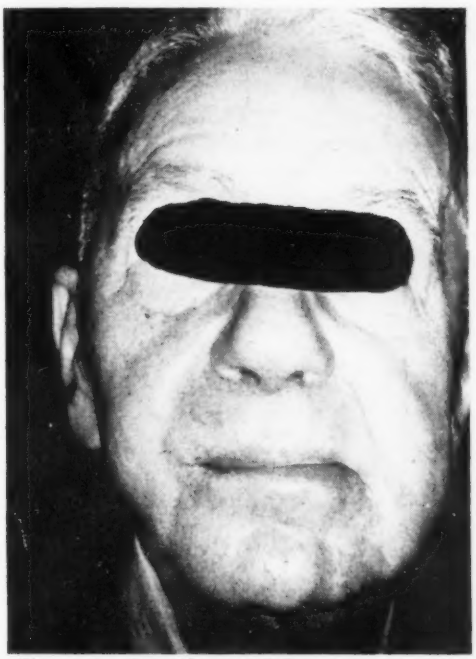

Figure 9.—Case No. 7, J.E.P.

Figure 10.—Case No. 7, J.E.P.

Case No. 7. J. E. P., white male age 71. Tongue lesion biopsied on December 28, 1950, with positive diagnosis of squamous carcinoma Grade 2. The lesion was a 2 cm. ulcer on left side of tongue at junction of oral and basilar portions.

X-ray therapy was given by external and intra-oral portals to maximum dosage in January and February of 1951. Radon seed were implanted on February 6, 1951.

In October 1951, biopsies revealed squamous carcinoma of left side of tongue and floor of mouth. The composite operation was carried out after preliminary lining graft was prepared after Edgerton's technique.

Figure 11.—Case No. 8, J.F.W.

Case No. 8. J. F. W., white male age 65. This patient presented an ulcerated lesion 2. 5 cm. overlying the left lower alveolar ridge. Biopsy revealed squamous carcinoma grade 2. Mandibular necrosis was visible in the intra-oral ulcer.

Irradiation had been given five years ago and again in 1949. There were no palpable nodes. Composite operation was carried out on June 28, 1950.

There is no evidence of recurrence at this time. The nutritional state is good. The patient is working and well adjusted.

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SURGERY IN DEAFNESS*

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Surgery in deafness encompasses all the surgical procedures, both prophylactic and therapeutic, performed on the organ of hearing and its adnexa, which will improve the existing level of hearing or prevent the further impairment of hearing. In the pre-antibiotic era, otologic surgery was primarily concerned with preservation of life and, as a consequence, little concern was given to conservation of hearing. However, with infection controlled today by specific drugs so that it is no longer a hazard to life, the surgery in otology must strive to preserve and restore the maximum amount of hearing function.

The surgical procedures which, I believe, have the greatest effect on preservation of hearing are:

1. The adenoidectomy.

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*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 30, 1952.

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2. The myringotomy.
3. The mastoidectomy, the type depending on the extent of disease and the existing level of hearing.
4. The fenestration operation.

THE ADENOIDS IN DEAFNESS

There are no structures adjacent to the ear more closely related to the function of hearing than the adenoids. Therefore the proper administration of therapy to the adenoids is vital to the management of deafness. Because of their proximity to the eustachian tube orifices, infection and hypertrophy can produce tubal obstruction, otitis media, and conductive deafness. It is generally conceded that adenoid hypertrophy is the most common cause of early deafness in children. Therefore, it is evident that whenever an impairment of hearing occurs in the presence of enlarged adenoids, the treatment is removal of those adenoids.

Removal of the adenoids is best accomplished by surgical means. Surgical removal is indicated whenever there is gross evidence of lymphoid tissue in the nasopharynx. Radium and external irradiation of the nasopharynx should be reserved for those cases in which there is no gross lymphoid tissue in the nasopharynx, yet the symptoms of eustachian tube blockage with otitis media continue.

I am convinced that the proper adenoidectomy, as emphasized by Meltzer,[1] should be one meticulously performed under direct vision, using the adenotome for removing the bulk of tissue and employing sharp punches for removing completely the lymphoid tissue around the eustachian tube orifices, in the fossa of Rosenmuller, inferiorly along the salpingopharyngeal folds and scattered in patches over the oropharynx.

When the lymphoid tissue is removed thoroughly and completely in this manner, uniformly good results will follow and the danger to hearing from this cause will be eliminated.

MYRINGOTOMY

Myringotomy is one of the simplest and most fundamental surgical measures in the prevention of deafness and should be performed unhesitatingly whenever the signs and symptoms of fluid or abscess in the middle ear are present.

With the widespread use of antibiotics today, there is a growing tendency among clinicians never to do myringotomies in acute suppurative otitis media but instead, to sterilize the infection with massive doses of drugs. I have seen this work in a surprising number of patients and the infectious process has been reversed without sequelae. However, in a small percentage of these cases, irreparable damage to hearing will ensue because of an unresolved exudate remaining in the middle ear causing adhesions to occur, which will immobilize the drum and ossicles impeding function and impairing hearing.

Hoople[2] has noticed a 20 per cent increase in the incidence of fluid in the middle ear since the advent of biochemical treatment of purulent otitis media and the greater part of this increase was in children.

Myringotomy in acute suppurative otitis media should be carefully placed in the posterior inferior aspect of the drum and wide enough to afford sufficient drainage.

Myringotomy for serous otitis media should be only large enough to remove the fluid and can be placed in the posterior inferior or the anterior inferior quadrant of the ear drum. Hearing function is best conserved when drainage is established early for fluid in the middle ear.

SIMPLE MASTOIDECTOMY

The incidence of mastoidectomy has decreased markedly today, but the operation is not extinct. It is still a necessary surgical procedure to eliminate resistant disease of the temporal bone. The type of mastoidectomy performed is determined by the extent of disease and the existing level of hearing.

The simple mastoidectomy offers the least disturbance to the mechanism of hearing and is performed when disease is limited to the mastoid bone, the mastoid antrum, and the aditus. It is indicated when recurring attacks of mastoiditis develop with impairment of hearing secondary to otitis media, even though chemotherapy has been employed. This mastoiditis may be mani-

fested by persistent mastoid tenderness (over the antrum, tip, and emissary vein) and accompanied by pain, headache, and aural discharge. A simple mastoidectomy is necessary also when the hearing is at a practical level, and, when, in the presence of an acute mastoiditis, labyrinthine symptoms or signs of facial weakness develop.

The simple mastoidectomy is best performed through an endaural approach. The mastoid cells are carefully and completely exenterated with a high speed, motor driven burr. The dural plate, lateral sinus plate, and the posterior bony wall of the external auditory canal are skeletonized. The mastoid antrum is opened, the aditus is widened, and the incudomalleolar joint is exposed.

Wide surgical drainage is afforded with this procedure; the middle ear is left undisturbed and hearing is conserved.

MODIFIED RADICAL MASTOIDECTOMY

The modified radical mastoidectomy is the procedure of choice in disease of the epitympanum, aditus and the mastoid process, when hearing is serviceable and the ear drum practically intact. There is usually a small perforation of Shrapnell's membrane or a marginal perforation at the posterior superior region of the drum. A cholesteatoma may or may not be present.

The operation entails exenterating the mastoid cells through an endaural incision and removing the posterior bony canal wall and the lateral wall of the epitympanic space, so as to visualize this space. The head of the malleus and the incus may or may not be removed, depending on the extent of their involvement. A tympanomeatal flap is laid over the ossicles and into the mastoid cavity, thus converting the epitympanic cavity and the mastoid cavity into one common space and sealing off the tympanic cavity below.

Maintenance of servicable hearing can be obtained with this type procedure in a large percentage of cases, even though the disease is usually an extensive process.

RADICAL MASTOIDECTOMY

In the radical mastoidectomy hearing is of no consequence, since it is always below any salvageable level; therefore, the primary consideration must be thorough elimination of disease with the attainment, postoperatively, of a dry, epithelialized mastoid cavity. In achieving this goal, I have followed certain broad principles, which, I think, are necessary:

1. Remove every vestige of disease from the mastoid and tympanic cavities, using the proper illumination, magnification, and endaural instruments.
2. Curette vigorously the osseous portion of the eustachian tube under direct vision removing all mucous membrane and the surrounding peritubal cells.
3. Place a split-thickness skin graft so as to completely cover the tympanomastoid cavity and pack in place for nine days without interference.

FENESTRATION OPERATION

In gradual, progressive deafness secondary to otosclerosis, the fenestration operation offers the best chance to restore permanent, serviceable hearing to a high percentage of cases.

The operation was made practical by Julius Lempert[3] in 1941, when he first described his one stage nov-ovalis technique for opening the surgical dome of the vestibular labyrinth. This created a new window to the inner ear, so that sound might by-pass the normal pathway which was impeded by fixation of the stapes due to overgrowth of bone.

The operation was hailed as one of the great advancements in otology and has in the past decade successfully restored hearing to nearly 20,000 persons who were hopelessly deaf. It is not perfected entirely, although rapid strides are being made toward that end. The biggest deterrent to the success of the operation has been closure of the newly created labyrinthine window. The incidence of this closure, which amounted to almost one half the cases in earlier days, now approximates less than 10 per cent, varying with the proficiency of the operator.

The fenestration operation requires an ingenious technique of exposing the epitympanic space and the horizontal semicir-

cular canal through an endaural approach. It is then necessary to remove the incus, head of the malleus, and the posterior bony wall of the external auditory canal and to make an opening approximately 2 by 5 mm. in the bony labyrinth over the ampulla of the horizontal semicircular canal. A tympanomeatal flap is laid over this newly created window. The flap hermetically seals the tympanic cavity and forms a functioning, protective cover over the exposed membranous labyrinth for transmission of sound.

With newer developments and refinements in this technique, the incidence of bony closure is being progressively decreased. These improvements have been outlined by Lempert[4,5] and include:

1. Making the fenestra as wide as possible in the surgical dome of the vestibule of the labyrinth.
2. Using the bone-dust-free technique.
3. Invaginating and securing in place the portion of the tympanomeatal flap over the fenestra, so that primary union of the skin and fenestra rim takes place.

It has been my experience that when this improved technique of fenestration surgery is meticulously performed in an organized manner with the necessary patience and thoroughness, a gratifying result will usually be forthcoming.

SUMMARY

The most important surgical procedures in the management of deafness are:

1. A thorough and complete adenoidectomy.
2. A well placed, early myringotomy.
3. A proper mastoidectomy, which will eradicate disease and preserve the greatest amount of hearing function.
4. A fenestration on selected cases of deafness secondary to otosclerosis.

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DISCUSSION

Dr. Miles L. Lewis, Jr. (New Orleans.) I would like to congratulate Dr. Tabb on his excellent and timely paper, and would like to make a few comments on the topic.

One point should be made clear: All surgery mentioned in Dr. Tabb's presentation, be it curative or preventive, deals only with conduction deafness, or the conduction component of mixed deafness. Thus far, there has been no successful medical or surgical treatment of nerve deafness.

With the advent of the sulfonamides and later the antibiotics, the incidence of complications of acute otitis media dropped to practically nothing, and with this drop, otological surgery and the interest in otology dropped also. Then, as Dr. Tabb stated, the interest in otology was necessarily directed toward the conservation and improvement of hearing. This interest in hearing received a great stimulus by the development of the one-stage fenestration operation by Dr. Lempert, and to a lesser degree by the work of Crowe and his coworkers in the treatment of conduction deafness in children due to eustachian tube obstruction by lymphoid hyperplasia. The careful adenoidectomy, followed when necessary by irradiation of the nasopharynx, is the most important surgical measure used in deafness, and its results are most gratifying.

I would like to emphasize Dr. Tabb's remarks about the desirability and importance of doing a myringotomy. Any time there is even moderate bulging of the of the tympanic membrane in acute otitis media a myringotomy should be done.

The simple mastoidectomy is the treatment of choice of masked mastoiditis, that is, acute mastoiditis, the signs and symptoms of which have been "masked" by inadequate therapy with antibiotics.

In the modified radical and radical mastoidectomies the primary concern is the conversion of a potentially dangerous condition, in which intracranial complications can occur, into a safe condition. The second consideration is the preservation or improvement of hearing.

Otosclerosis is a progressive disease with increasing deafness, and, as it progresses, is often complicated by secondary nerve degeneration. Only by early case finding can the patient be evaluated for fenestration surgery while he is still an ideal candidate, and the careful selection of ideal candidates is the most important single factor in obtaining good results from fenestration surgery.

WHAT THE GENERAL PRACTITIONER SHOULD KNOW ABOUT CROSSED EYES*

B. MORRIS PHILLIPS, M. D.

MONROE

Many questions arise in the minds of parents of the child whose eyes cross and it is usually the family doctor who is first called upon to answer these questions. The answers given may determine whether the child is to have a sound and useful eye or a blind one, not to mention the effect they may have on the personality of the child.

For one who is in a branch of medicine other than that of diseases and surgery of the eyes there is little reason to learn about crossed eyes. It is the purpose of this paper, to render information which may enable the practitioner to give sound advice to the naturally worried parents of the child with crossed eyes. Some of the most commonly asked questions as regards crossed eyes will be given and briefly the answers. The term crossed eyes or cross-eyed is used in a broad sense in this discussion and merely refers to eyes that are not straight.

One of the questions frequently asked first is "Why did the eyes of my child cross?" Of the eyes that are not straight at birth—and Scobee says that slightly more than one-fourth of all the children with crossed eyes have them from birth—inherited defects are the most frequent cause. Almost invariably this condition runs in families. Parents need not necessarily show the defect themselves to pass it on to their children. Birth injuries cause eyes to be crossed from birth. It has been estimated that 40 per cent of all babies born after a normal spontaneous delivery have multiple tiny bleeding points in the brain as a result of compression of the head during birth. In the great majority of instances these small hemorrhages are absorbed and no damage is done. Only about 1.5 per cent of all children in this country at the present time, it is said, have eyes that are not straight. The difference between 40 per cent and 1.5 per cent is great but it is not difficult to see, however, that some disturbance of one of the delicate control centers in the brain might do permanent damage and thus result in crossed eyes.

A common age at which eyes are noted to cross is between two and one-half to three years. Convergence and accommodation are so closely allied that when accommodation is excessive, convergence tends to be excessive too, and if fusion is weak, the eyes may actually cross. Careful studies have shown that in the majority of children whose eyes cross between the ages of two and one-half to three years, there will be found some abnormality in the muscles or attachments of the muscles that move the eyes as well as the far-sightedness. Many childhood diseases occur at or about this same age and the parents may blame the most recent illness when the eyes finally do cross. Actually, the diesase may have hastened the time of crossing but it probably did not cause the eyes to cross all by itself. Diphtheria is one disease which may affect the muscle directly.

Eyes cross after injury and blinding of one eye. Fusion is now impossible and the blinded eye will deviate in one direction or the other since there is no reason for the two eyes to work together as a team.

In summary, eyes may cross because of inherited abnormalities of the muscles that move the eyes, because of far-sightedness, because of injury to the brain or to the eye muscles at birth, because of loss of vision in one eye, or because of some disease which affects the eye muscles directly or portions of the brain which control the eye muscles or movements of the eyes.

"Will a child outgrow crossed eyes? If so, how long will it take?" About the only type of crossed eyes which is ever outgrown is that type which is almost purely nervous in origin. All other types are permanent unless treated. Thus, the parents of cross-eyed children should beware not only of the advice but also of the source of that advice when they are told that the child will probably outgrow its crossed eyes. Parents who

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, April, 1952, Shreveport, Louisiana.

wait to see if their child will outgrow crossed eyes expose him to psychic trauma to the extent that by the time he reaches kindergarten his behavior is already affected. By the time he reaches the first grade there are definite psychologic scars on the personality. He feels different from other children. He has the feeling of not belonging.

"Why is it important to straighten a child's eyes before he or she starts to school?" The psychologic scars on the personality can be avoided entirely if the eyes are straightened early in life. This allows the child a normal development with straight eyes in the society of his or her fellows—a normal development because the child need no longer feel different from the others. Eyes which are crossed at birth are not going to straighten with the passage of time. Therefore treatment should be begun at an early age. Its too late to wait until a child is old enough to decide whether he or she wants the eyes straightened, and occasionally, one encounters the parents whose stated reaction is just that. One has but to listen to the bitter remarks of adults with crossed eyes whose parents never did anything about the situation to realize that the child, when grown, is not in the least grateful for such treatment.

"Why not wait until ten or twelve years of age to have the eyes straightened?" Crossed eyes not only affect the personality of the patient but the eyes as well. There are two main types of crossed eyes, the monocular type and the alternating type. If the former is not corrected early blindness of disuse and abnormal retinal correspondence may develop. In the alternating type the bad habit of alternating suppression may develop. Any of these occurrences may prevent the eyes from working together as a team and act as an obstacle in reaching the goal of fusion. Even if the eyes are straightened by operation there is no guarantee that they will remain straight unless fusion is attained. The best time for treatment is early.

"Why is it necessary to dilate the eyes of the child with crossed eyes when examining for glasses?" Briefly, this puts the accommodation or focusing power of the eye at rest. If the eyes are crossing due to excessive far-sightedness then this allows the amount of far-sightedness to be measured fairly accurately and thus obtain the correct prescription for glasses.

"Why do glasses seem to straighten, at least partially, the crossed eyes of some children?" If the eyes are crossed only because of far-sightedness, then glasses will completely straighten them. If they are not completely straightened with glasses then obviously something more than glasses alone is needed to get the eyes straight. This usually means an operation and exercises.

"Does the child with crossed eyes need to wear glasses all the time?" The answer is yes. If a child has crossed eyes due to far-sightedness and wears glasses to correct this then removing the glasses for even a few minutes causes the child to use his ciliary muscle excessively to overcome his far-sightedness. This causes overconvergence and crossing of the eyes again.

"Must a child, whose eyes cross, necessarily wear glasses all of his life?" This question can only be answered by replying, maybe. Some children outgrow a part, and occasionally, all of their far-sightedness. If they outgrow it entirely then certainly there will be no need for glasses. If, on the other hand, they outgrow only a part of it and that part which remains is still sufficient to make the eyes cross without correcting glasses, then glasses should be worn as long as they keep the eyes straight. If fusion can be developed and the patient is only moderately far-sighted then it may be that glasses need be worn only for reading and close work.

Often parents ask if glasses correcting far-sightedness cannot be removed after an operation to straighten crossed eyes. The operation will have absolutely no effect on the amount of the far-sightedness. It is aimed at correcting the position of the eyes only. The child will be just as far-sighted after the operation as before it. Therefore, there will be just as much need for glasses

to correct the far-sightedness after the operation as before. If a child is very slightly or even moderately far-sighted, often the glasses may be removed after an operation only if fusion has developed. If the child is very far-sighted, he would need glasses whether the eyes were crossed or not and will continue to need glasses after an operation.

"Why do glasses which are prescribed for far-sightedness often blur vision?" Glasses take the load off the ciliary muscle in the far-sighted person, but since this muscle has had overwork for years it is not going to relax overnight and let the glasses do the focusing. It takes from two days to two weeks for this, depending upon how badly the person needed the glasses in the first place. Until the ciliary muscle does learn to relax, the person is made near-sighted and thus distant objects become blurred. When the ciliary muscle relaxes and allows the glasses to do the work which they were intended to do then objects are seen clearly at a distance.

"How often is it necessary to change the glasses of a child with crossed eyes?" A far-sighted child whose eyes cross, whether the glasses straighten the eyes partially or completely, should have his eyes examined at intervals of at least every six months. This does not necessarily mean that the glasses are changed every six months but it means examining the eyes to see whether or not the glasses do need changing. The far-sighted child tends to outgrow some of his far-sightedness because his eyes are small. If the eye grows the far-sightedness becomes less. Glasses which correct the far-sightedness today may be too strong six months from now because the child will have grown some, his eyes will have grown some and his far-sightedness will have decreased some.

Often the question is asked, "Do people whose eyes cross see double?" The answer is that they do not. The reason they do not see double is that their brain has learned to ignore completely the image coming from the crossing eye. A child whose eyes cross undoubtedly sees double for a short time but the situation is so confusing and uncomfortable that the brain quickly learns the trick of ignoring or suppressing images coming from the crossing eye. This is known as suppression and the unused eye suffers from lack of use.

"Why is it necessary for children with crossed eyes to wear a patch over one eye for several months and what does it accomplish?" A conditioned reflex is a reflex, something done automatically, which occurs every time the conditioning stimulus is repeated. The blindness of disuse in a crossing eye is a conditioned reflex and the conditioning stimulus for this developing blindness in the crossing eye is looking at things with the good eye. To break this conditioned reflex the good eye must not be used at all. Patching the good eye accomplishes two things: (1) It does away with the conditioned reflex which made the crossing eye lose vision in the beginning; and (2) it strengthens vision in the crossing eye by forcing its use.

Patching the good eye a few hours a day is only a waste of time. It must be patched from the moment the child awakens in the morning until he goes to sleep at night. This is the only way in which the blindness of disuse may be overcome.

"What are eye exercises?" Orthoptics is the medical term for eye exercises. When eye exercises are prescribed, it means exercises which will help the patient along toward the ultimate goal of two straight eyes working together as a team. Eye exercises are of no value whatever unless they are carried out in such a fashion that the eyes learn to see together as a team. Orthoptic exercises have two goals: First, the destruction of abnormal retinal correspondence, and second, the development of fusion. Unless fusion powers can be built up to a normal level, there is no guarantee that the eyes will remain straight even after operation.

"Is an operation for crossed eyes dangerous and will an operation on one eye bother the sight of the other, and will it improve the vision of the eye operated on?" The sur-

geon who assures the patient that there is absolutely no danger in an operation for crossed eyes is not quite correct. There is a risk in any operation, but in the case of the one for crossed eyes, it is very small. Children are usually operated upon under general anesthesia. Adults are frequently operated upon under local anesthesia. The operation will not affect the vision in the eye operated on nor will it affect that of the unoperated eye.

"Are both eyes ever operated on at the same time?" It is sometimes necessary to operate on both eyes at the same operation, although only one eye is ever crossed at any one time. It is seldom possible to straighten crossed eyes which are markedly crossed in a single operation or by operating on just one eye.

"How early in life can the treatment of crossed eyes be started?" The treatment of crossed eyes, without exception, should begin as soon as the eyes are noted to cross. In the infant this usually amounts to nothing more than the alternate patching of first one eye then the other in order to prevent the development of the "blindness" of disuse. In the young child the treatment is usually the prescription of glasses and the patching of the crossing eye. If surgery is needed, a child should have the benefit of an operation as soon as an adequate diagnosis can be made. This is usually around three or four years of age. If at all possible, the eyes should be straightened before the child is five and one-half to six, when his fusion powers would normally reach their full development. Other reasons have already been enumerated for straightening the eyes early.

Eyes are a team, a most remarkable team. It is entirely possible to keep this team working properly in the vast majority of children if they are intelligently cared for.

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OTITIS EXTERNA*

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Otitis externa is one of the most common diseases which brings patients to the otorhinolaryngologist and comprises from 5 per cent to 40 per cent of their practice. The anatomical pathologist seldom sees these patients as external otitis is rarely fatal or terminal and only occasionally is a biopsy taken. On the other hand, the clinical pathologist, if he develops interest in the condition, can, through cooperation with the otorhinolaryngologist or the physician treating the patient, aid greatly in the specific diagnosis of this chronic infection and determine specific therapy.

These inflammatory conditions of the external canal are usually classified by their clinical signs and symptoms (Table 1).[1] The etiologic agent, on the other hand, is often ignored; yet one organism can produce furunculosis, cellulitis, or perichondritis. The cure for otitis externa is the elimination of that specific organism. Improper treatment results in further excoriation, exudation, and secondary infection.

The external ear is covered by squamous epithelium in its entire membranous, cartilagenous, and osseous portion and tympanic membrane. The pH is acid. Bacteria and fungi may remain dormant in the osseous portion but some species like *Pseudomonas aeruginosa* are never found in normal ears.

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 30, 1952.

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TABLE 1
CLASSIFICATION OF EXTERNAL OTITIS

A. Acute inflammation
   1. Furunculosis
   2. Cellulitis
   3. Erysipelas
   4. Perichondritis
   5. Thrush
   6. Impetigo
B. Chronic inflammation
   1. Acne
   2. Cellulitis
      (a) Mycotic infections
         (1) Actinomycosis
         (2) Coccidioidomycosis
         (3) Blastomycosis
         (4) Cryptococcosis
         (5) Moniliasis
         (6) Aspergillosis
         (7) Penicilliosis
         (8) Mucormycosis
         (9) Epidermomycosis
   3. Perichonditis
   4. Parasitic organisms
C. Chronic specific inflammation
   1. Syphilis
   2. Tuberculosis
   3. Sarcoidosis
   4. Leprosy
   5. Leishmaniasis
   6. Frambesia
   7. Tularemia

Any physical agent that destroys the intact epithelium or changes the pH of the osseous portion may cause the dormant organisms, or organisms introduced at the time of trauma, to produce external otitis. The first stage is inflammation, swelling, exudation and pain, followed by necrosis and suppuration. The entire external canal may be involved and spread by excoriation to the auricle.

ISOLATION OF CAUSATIVE AGENT

The causative organism should be isolated. The recovery of this organism in pure culture is practically impossible if the external canal is simply swabbed out. A simple technique developed by McLaurin will produce pure cultures in the majority of cases.[2] The method is as follows: A quick scrub detergent is used to clean the hands. Sterile cotton is wrapped around the end of a metal applicator. The greatest quantity of the exudate and debris is removed from the external canal by using suction with a Frasier tip. The attic and tympanic membrane is cleaned by suction using a malleable McLaurin aspirator. The cotton tipped applicator is then gently placed in the canal and the remaining exudate in the osseous portion removed. The culture media is immediately streaked with this exudate at the time of removal. This technique is relatively non-painful, is easy, and pure cultures can be expected. Our laboratory supplies a stoppered glass tube with a slant of brain heart infusion agar for this type culture. This media will support the growth of a majority of organisms. It is easily made and the cork or rubber stopper keeps the media moist.

The causative organism is then identified by routine bacteriological and mycological methods. A sensitivity plate is set up as soon as growth is obtained using the disc type of sensitivity testing. Since the external ear is readily available to therapy and any concentration of specific medication can be instilled, we use the higher concentration discs in our sensitivity tests. All the familiar antibiotics and chemotherapeutic agents are tested including a few of the proprietory drugs. The latter drugs are dropped on blotter paper discs that have previously been cut. They are placed on the media desired. The sensitivity plates are read in twelve to eighteen hours. Specific treatment can be instituted while the cultures are being identified.

In a brief series of cases of external otitis we have recovered the organisms shown in Table 2. Their reaction to the an-

TABLE 2
ETIOLOGIC AGENT IN OTITIS EXTERNA

Bacteria:	
Micrococcus pyogenes, var. aureus	62
Paracolon bacilli	12
Aerobacter aerogenes	10
Streptococcus pyogenes	10
Pseudomonas aeruginosa	8
Alcaligenes fecalis	4
Klebsiella pneumoniae	2
Proteus vulgaris	2
Proteus mirabilis	2
Diphtheroids	1
Fungi:	
Aspergillus niger	2
Mucor mucedo	2
Fenicillum spinulosum	2
Total:	118

tibiotics were similar to those found by other investigators. There have been several cases in which the organism recovered was not sensitive to any antibiotic a self respecting member of that group should be, but it was sensitive to an antibiotic or chemotherapeutic agent that ordinarily is considered of little value in treating that organism. The use of the indicated therapeutic agent gave prompt healing of the inflammation.

TREATMENT

Treatment in brief consists of two factors: (1) keeping the external canal dry, and (2) applying the proper therapeutic agent in the correct concentration. The latter is determined by the sensitivity tests. The former is accomplished by irrigating the ear with any nonirritating solution of a neutral or acid pH. Tap water is satisfactory if the canal is left dry. The canal is then kept dry by the p. r. n. insertion throughout the length of the canal of a long dry absorbent cotton wick. These wicks are rolled by hand and inserted into the canal by the aid of a tooth pick.

Using this method of treatment, external otitis is rapidly cured, as a specific therapeutic agent is placed in contact with the causative organism, and the canal is kept dry of excoriating and macerating exudates.

CONCLUSIONS

1. External otitis is most often a bacterial infection and a single organism is the etiologic agent.

2. The exudate to be cultured must be obtained carefully and be streaked immediately on the media.

3. Sensitivity of the causative organism is easily determined and regulates therapy.

4. The external canal must be kept clean and dry at all times.

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SPONTANEOUS RUPTURE OF THE ESOPHAGUS: CASE REPORT*

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SHREVEPORT

Spontaneous rupture of the esophagus was first reported in 1724, by Boerhaave, a Dutch physician. Until 1946, there had been only 50 cases reported. Barrett, in reviewing the literature, in 1946, noted that none had survived. In 1947, Barrett reported a case that was successfully treated by operation. Since this report there have been a number of recoveries following early diagnosis and operation.

ETIOLOGY

The etiology of this condition is not known. In cases reported several factors that may produce the condition have been noted. An increase of intraesophageal pressure, brought on by forceful vomiting, straining, or external trauma to the abdomen or thorax has been observed in all the cases studied. Beal, in 1949, reported a case due to violent coughing.

The rupture of the esophagus usually occurs in the lower one-third. Studies have shown that the esophagus is not as strong structurally in this region as in the middle and upper portions. Microscopic studies of the ruptured portion do not reveal any pathological lesions about the site of the rupture. After the esophagus ruptures there is a resulting mediastinitis and pleuritis.

SYMPTOMS

The symptoms produced by the rupture are often confusing. The patient presents the picture of acute shock that could be caused by either an intrathoracic or upper abdominal lesion. Some cases have the appearance of an acute coronary occlusion. Others appear to simulate a ruptured peptic ulcer and several cases reported were operated on for an abdominal condition before the esophageal lesion was discovered. However, there is a triad of symptoms that have been fairly constant in all of these cases studied. There are (1) rapid respira-

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 30, 1952.

tion, (2) rigidity of the abdomen, (3) crepitus in the neck.

TREATMENT

The treatment of ruptured esophagus is early operation. Although the patient is in acute shock, operation should not be delayed as all the cases that have recovered were operated on as soon as possible after the diagnosis had been established.

CASE REPORT

As far as we know there has not been a case of spontaneous rupture of the esophagus reported from Shreveport. As these cases are not seen very often, and too, as there has been considerable investigation of esophageal lesions in the last few years we thought it would be well to report an additional case.

F. P. C., white male, 37 years of age, was transferred to the Caddo Parish jail from a neighboring parish. He had been confined to jail for being drunk. He was extremely violent and before leaving town had been given a hypodermic injection, probably morphine. It took six men to hold him and put handcuffs on him. He broke the cuffs on the way to Shreveport. He arrived at the jail at 1:20 P. M., September 10, 1950. As he was very violent he was placed in solitary confinement. He continued to be very violent and raved until about midnight, at which time the trusties state that he became quiet. Between 5:30 and 6:30 A. M., one of the trusties looked in the cell and saw him lying motionless on the floor. The jailer was notified and upon entering the cell found him dead. He was found lying face down. There was a large amount of vomitus on the floor about the body. Several blood clots were noted in the vomitus. Upon turning the body over, there was marked cyanosis of the head, face, and upper chest.

The body was taken to the morgue and an autopsy was performed. The autopsy revealed a white male, six feet tall, weighing about 200 pounds. Rigor mortis was complete. There was marked lividity over the chest, neck, and face. Both eyes were congested and there was hemorrhage into the anterior chamber of the left eye. Upon opening the chest cavity, in the left pleural cavity, there were approximately 300 cc. of bile stained fluid. Both lungs were partially collapsed. On sectioning, the left lung showed a large amount of thin aspirated mucus filling the main stem bronchus and scattered throughout the bronchial tree. This mucus was sufficient to cause obstruction. There was no consolidation, infarct or atelectasis. The right lung showed a similar condition but to a less marked degree. The esophagus was exposed and examined. Beginning just above the opening in the diaphragm, there was a longitudinal tear, extending 5 cm. upwards on the anterior surface. The edges of the wound were slightly jagged. Pressure on the stomach was followed by an escape of fluid through the opening in the esophagus. The wall of the esophagus was very thin in this area but did not show any gross pathology. There were no ulcerations or varicosities. The heart was normal in size. The myocardium showed gross evidence of myocarditis. On sectioning, the liver showed fatty changes without cirrhosis. The stomach was dilated and contained about 200 cc. of bile stained fluid. The rugae were flattened. There were numerous petechial hemorrhages and ecchymotic areas of the mucosa. There were no ulceratons noticed. Both kidneys showed marked congestion.

Microscopic studies were made of the liver, heart and kidneys:

Liver — slight cirrhosis with marked fatty changes. Architecture destroyed, changes probably due to alcohol.

Heart—Chronic myocarditis.

Kidneys—Hemorrhagic glomerular nephritis.

Specimens of the stomach contents, blood and urine were taken for alcohol analysis:

Blood alcohol	0.10 per cent
Stomach alcohol	0.07 per cent
Urine alcohol	0.08 per cent

COMMENT

This man was a known alcoholic for twenty years. The history shows that he was extremely violent. It is our opinion that while confined to the jail he became nauseated and vomited on several different occasions. The forceful vomiting caused a spontaneous rupture of the esophagus. The findings of the ruptured esophagus follows several previous cases reported in which it had been noted that these patients had been on a protracted drinking spell, had become nauseated, and had vomited violently. It is our opinion that this death was due to the aspiration of mucus and vomitus into the lungs following spontaneous rupture of the esophagus.

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CREEPING ERUPTION (LARVA MIGRANS)* AT KEESLER AIR FORCE BASE

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Since the classical presentation of creeping eruption was made, in 1926, by Kirby-Smith, White and Dove,[1] I will begin my discussion with their description of this disease.

Creeping eruption is characterized by a linear, tortuous, and serpiginous eruption caused by the migration of a nematode parasite within the skin and accompanied by intense itching. The most recent visible lesion is a very narrow erythematous formation along the course traversed by the worm. Soon a slightly raised line representing the location of the burrow can be palpated. This line becomes visibly elevated, more or less continuous and vesicular. Sometimes bullae are formed. The surface of the lesion dries, resulting in a thin crust. When the parasite travels it moves from a fraction of an inch to several inches a day, advancing, as a rule, more rapidly at night.

Following the penetration of the skin the parasite apparently remains at the point of entry before migrating, resulting in a reddish, sensitive papule not unlike that of the bites of chiggers. From these points the advancing threadlike burrows may be observed within from two to four days. The raised portion of the skin over the burrow has the appearance of that made by a mole in meadows and elsewhere. The end most distant from the parasite may be 0.5 cm. in width and may sometimes give the lesion a tapering appearance. Abrupt changes in the direction of the burrow are frequently observed. The migration has been observed on a number of occasions to have continued for several weeks, and some observations indicate that the larval migration is interrupted by periods of rest. Often the scratching results in bacterial infections and raw surfaces. The lesions can occur on every part of the body; however, since infections take place on those portions coming in contact with the soil, the lesions occur most often on the feet, buttocks, and hands.

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 30, 1952.

PRESENCE OF THE DISEASE ON THE MISSISSIPPI GULF COAST

At Keesler Air Force Base we saw 22 cases of creeping eruption during the year 1951. This base is located on the Mississippi Gulf Coast at Biloxi. In a review of the literature we found no report of creeping eruption in the region of the Gulf Coast of Mississippi. Several physicians in this area, including the dermatologist, reported that they see creeping eruption in their practice.

Although exact figures cannot be given there are approximately 45,000 individuals who are eligible for treatment in our dispensaries and clinics. This includes military personnel and their dependents. Because these people are prompt to come to us for medical attention, we feel that we saw almost every case of creeping eruption that occurred in this group. This is interesting because it should give a good index to the incidence of the disease per 1000 people in the area. From the above we can estimate that annually there is approximately one case of creeping eruption per 2,000 persons on the Mississippi gulf coast.

DIAGNOSIS

The disease presents itself in the classical textbook description in a very high percentage of the cases; therefore, the diagnosis of creeping eruption is usually not difficult. No other disease can produce the typical serpiginous burrow that day by day advances from one end. Itching, almost invariably present, is usually intense. The only difficulty in diagnosis is that many doctors have never seen the disease and a diagnosis of creeping eruption is never considered.

However, a small percentage of the cases can be diagnosed only by the most careful observer. These fall into three groups: (1) vesicular and bullous, (2) urticarial, (3) erythematous. The most common case of this type is one in which the onset is acute

and the chief manifestation is one of vesiculation and bullae formation. These can easily be mistaken for a dermatitis venenata and possibly for a bullous erythema multiforme or bullous drug eruption. Usually an occasional burrow can be found that will make the diagnosis evident. Another type to look for is one in which urticarial lesions develop at the site of entrance of the larvae. The urticarial response seems to inhibit the motility of the larva and burrows may be minimal or absent. An erroneous diagnosis of urticaria, insect bites or ground itch may be made. This clinical picture is seen when the patient reports promptly for treatment and has had mulple larvae penetrate the skin. Burks[2] states that he has seen cases of generalized urticaria produced by an allergic response of the body to the presence of one or more larvae.

The erythematous type may have only a deep erythema at the site of entrance of the larva. Rigdon[3] describes a typical lesion of this type in discussion of his daughter's case. A positive diagnosis of creeping eruption cannot be made until a typical burrow is found at some other site or later in this lesion.

A fourth type of creeping eruption case in which the diagnosis is sometimes difficult may occur in any of the above disease patterns. These are cases in which excoriations, crusts, pyoderma, cellutitis, or overtreatment obliterate the normal appearance of the disease. Fortunately the percentage of the atypical cases is small.

ETIOLOGY AND INCIDENCE

The larvae *Ancylostoma brazileinse* and *A. caninum*, the dog and cat hookworm, have been proven to be the causative parasites in this region. Kirby-Smith, White and Dove,[1] White and Dove,[5] and Shelmire,[6] did excellent work in demonstrating both clinically and experimentally that this larva produced creeping eruption. It is 1/50 of an inch long and 1/1000 of an inch wide.

The Southern endemic area begins in Florida and extends along the coastal states as far north as Maryland or New Jersey and as far west as Texas. The only inland states reporting cases of creeping eruption in significant numbers are Oklahoma and Arkansas. Occasional cases are reported from other states. The incidence is highest in Florida.[17] The other states with a high incidence are the Carolinas and Georgia.

Creeping eruption, which is world-wide in distribution, is not caused by this larva alone. Other larva that are reported to produce the disease with some degree of frequency are *Gnathostoma* (pig and cat nematode), *Gastrophilus* (botfly), and *Hypoderma* (warble fly grub). Characteristically, when one of these larva is producing the disease there is only one penetration, one track, and one larva. Multiple lesions and penetrations are not seen. Occasional cases are caused by the above larvae and these are usually in widely separated areas. Cases due to *Ancylostoma braziliense* have been reported from North America, Central America, South America, Africa, India, the East Indies and the Philippines.

HISTOPATHOLOGY

Histopathologically the larva and burrow are found in the prickle-cell layer. The larva is usually coiled and the burrow contains many swollen degenerating epithelial cells. In the corium there is some perivascular infiltration of lymphocytes and eosinophils.[7, 1]

TREATMENT

The treatment of choice has not definitely been established. The question to be decided is if systemic treatment is superior to topical applications. To most observers there is only one topical treatment that can be considered as satisfactory—freezing with ethyl chloride or carbon dioxide snow. Shelmire was the first to advocate this type of therapy and it has withstood the test of time.

Various other forms of topical therapy reportedly used with success have been as follows: Injections of phenol, iodine, chloroform, benzene, etc.; applications of tincture of iodine followed by ammoniated mercury ointment; onion poultices; electro-dessication; x-ray; incision followed by the application of a cauterizing solution such as phenol or nitric acid; and excision of the le-

sion. There have been reports recommending the use of each of the above methods; however, for one reason or another they have not proven to be as satisfactory as freezing with ethyl chloride. Most of them were either painful, produced a severe reaction, were disfiguring, or were ineffectual.

Even the most ardent advocates of topical treatment with ethyl chloride admit that it is not satisfactory treatment when scores of lesions are present. Therefore, a good drug for systemic treatment of this disease would be welcome. On reviewing the literature we found that fuadin, an antimony compound, had been recommended by Smith,[8] Wilson,[9] Rubin,[10] and Dolce and Franklin.[11] However, after reading far less favorable reports by Blank,[12] Hitch,[13] and Hailey,[14] we decided that this drug must not be exceptionally beneficial.

Stibanose, a new antimony compound, was reported by Wilson,[19] to be the most satisfactory treatment that he had found. We have not used this drug, but I am certain that it warrants clinical investigation.

Recent reports by Loewenthal[15] and van de Erve[16] were favorable to the possibility that hetrazan (diethylcarbomazine) might be specific for this disease. Loewenthal used a dosage of 2 mgm. per kg. three times a day and cured 7 of 8 cases in two to three weeks of therapy.

Using ethyl chloride freezing at three to five day intervals, we satisfactorily cured 8 cases with from one to five treatments. Some of these had more than one larva, but none had more than six. All patients were cured in less than four weeks. We were next faced with a man who had approximately one hundred lesions. Freezing with ethyl chloride was impracticable. At this time we obtained a small supply of hetrazan and he was given 150 mgm. three times daily. When there was no improvement in two weeks we increased the dosage to 150 mgm. four times daily. After another week there was still no improvement and the patient had become quite disturbed over the condition. Although continuing hetrazan, we began freezing some of the lesions with carbon dioxide snow at forty-eight hour intervals. In two weeks all of the lesions were inactive. The patient was not impressed with the role hetrazan played in his cure.

During this period we treated 5 other patients with the hetrazan, but only in conjunction with ethyl chloride freezing. Although we had no definite control group, the disease course apparently was not shortened by adding hetrazan to the freezing method. Therefore, we prescribed no more hetrazan and continued the freezing. Results were consistently good.

We wish to state that with our limited experience using hetrazan we were not trying to evaluate its effectiveness. We were only thinking of curing our patients speedily and safely. Etteldorf and Crawford[18] report that hetrazan can be given without toxicity in dosages up to five times as great as the 2 mgm. per kilogram three times daily that we used. Loewenthal[15] after curing 7 of the 8 cases in three weeks with 2 mgm. per kilogram thrice daily suggested that better results might be obtained by using 6 mgm. per kilogram. We will give hetrazan a more thorough trial at some future date.

SUMMARY

1. Cases of creeping eruption are present in significant numbers on the Mississippi Gulf Coast.

2. The diagnosis, incidence, etiology, histopathology, and therapy are reviewed and discussed.

3. Freezing with ethyl chloride is still the treatment of choice.

4. Hetrazan or stibanose may or may not prove to be specific systemic therapy.

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DISCUSSION

Dr. James W. Burks, Jr., (Gulfport, Miss.). Military medicine, through its study of large numbers of individuals from all parts of the United States congregated in rural sites, has made many noteworthy contributions in the field of Public Health. Dr. Knox is to be congratulated on his excellent and timely contribution.

Westward spread of this disease has become apparent to me in my practice on the Mississippi Gulf Coast where from mid-May to October each year I see the disease in increasing numbers. Heretofore, this disease was considered a Florida problem. Until recently, creeping eruption was seldom seen by the New Orleans dermatologist. For instance from 1944 through 1950 five cases came through my office: three had been in Florida for vacations; two had acquired the disease in Harvey, Louisiana. In 1951, numerous cases were seen by New Orleans dermatologists.

The Public Health aspects of this disease become apparent when we view the new twenty-six mile sand beach in the rapidly growing resort area of the Mississippi Gulf Coast.

CUTANEOUS BLASTOMYCOSIS*

A REPORT OF FIFTY-EIGHT UNPUBLISHED CASES

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American blastomycosis is a distinct clinical entity caused by a specific etiological agent—*Blastomyces dermatitidis*. It is one of the most formidable of the fungus infections and is also referred to as North American blastomycosis, oidiomycosis, Gilchrist disease and blastomycetic dermatitis. This disease falls into the group of diseases commonly referred to as infectious granulomas. The general term "blastomycosis" at once suggests lesions produced by blastomycetes, or budding fungi

Blastmycosis, like moniliasis, may be divided into two types: (a) localized or cutaneous and (b) systemic. The latter type usually has a grave prognosis and is characterized by pulmonary involvement and widespread distribution in oral cavity,[1] bones,[2-4] joints, internal organs,[5-7] and the central nervous system.[8-10] The cutaneous form is manifest as a subacute ulcerating process.

CLINICAL PICTURE

The clinical picture of cutaneous blastomycosis (Gilchrist) is the occurrence of a papulo-pustule which crusts and by peripheral spread forms an elevated, sharply marginated, verrucous and crusted patch. Removal of crusts reveals a pus-infiltrated base covered by irregular papillary elevations. These papillary growths are frequently very vascular and bleed easily with only slight trauma. Occasionally the base of the lesion may resemble an ordinary ulcer with exuberant granulations.

Perhaps the most typical and diagnostically significant characteristic is the violaceous, or purplish red, smooth, sloping border which is pin-pointed by miliary abscesses that appear as yellow points. The number of these small abscesses is related directly to the activity of the lesion. The lesions usually grow very slowly. The clinical course is irregular, but tends to chronicity.

Autoinoculation is thought to occur and in many patients multiple lesions are present. The subjective complaints vary. Except in those areas which are acutely inflamed or secondarily infected, pain is usually slight or absent. When healing occurs, either spontaneously or as a result of treatment, there is a gradual flattening and disappearance of the papilliform elevations by absorption, desiccation, and exfoliation. The seropurulent exudate diminishes and the

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*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 30, 1952.

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base of the lesion is replaced by a hypertrophic scar, later becoming soft, atrophic and pinkish white.

Multiple subcutaneous nodules are a more common and characteristic finding of systemic Gilchrist disease. These lesions are usually deep seated, but as they enlarge they approach the surface. Eventually, they soften and break down forming crusted and discharging ulcers or fistulous tracts leading to deeper parts of the subcutaneous tissue, muscle or bones. However, subcutaneous nodules and abscess may be seen in cutaneous blastomycosis and are referred to as of the gummatous type.

As indicated, the primary cutaneous disease follows a benign and subacute or chronic course. Only very rarely does systemic dissemination occur. Neither does it tend to spread to the regional lymphatics. The most frequently involved areas are the exposed parts—face, hands, wrists and forearms.

Man presumably acquires his infection from some outside source, and although proof is lacking, the natural host is apparently in nature. Most texts speculate with reference to soil, flora and fauna. That the infecting fungus appears in nature as a saprophyte has been suspected. Excavations, surface soil or grasses, thorns and branches of plants, dark cellars, rotting wood, and intimate contact with animals or carcasses have been suggested as sources of infection. Stober advanced the theory that the natural habitat was on damp rotting wood. He supported his contention by the isolation of a mold from wood in the living quarters of one of his patients. Three instances of infection in the dog have been reported.[11-12]

The disease is not considered contagious, but there are reports of its acquisition by long contact with an infected person, and two cases are reported in brothers. The only proven case of transmission from person to person was in a physician who developed a lesion at the site of a puncture wound received while performing an autopsy.[13]

The infectious nature has been shown by successful inoculation of animals.[14-16] Experimental intraperitoneal infection in the guinea pig, and a rapidly fatal systemic infection in mice have been produced. Attempts to produce cutaneous blastomycosis in mice have been unsuccessful.[17] The production of lesions on the chorio-allantoic membrane of the chick embryo by inoculation with the yeast form has been reported.[18] Earlier Goodpasture[19] had mentioned the susceptibility of the chick embryo to certain fungi.

It has been stated that there is no relation between the disease and sex, nativity, occupation or habits of the affected persons.[20] However, others consider it a possible occupational hazard in agriculturists, cattle handlers, florists, and mill and grain workers.[21] Many cases have a history of antecedent trauma which may be contributory. The differential diagnosis includes cancer, late syphilis, tuberculosis, granuloma inguinale, bromoderma, ioderma, and other deep fungus infections and other granulomas.

DIAGNOSIS

The diagnosis can be established by the direct microscopic demonstration of the *B. dermatitidis* in the miliary abscesses, biopsy examination revealing the organism, by culture, or by animal inoculation. The diagnosis cannot be confirmed without the demonstration of the organism, although perhaps either a positive complement fixation test or skin test to blastomyces vaccine may justify a presumptive diagnosis. Excision for biopsy is not contraindicated.

The histopathologic diagnosis of the disease with its etiological agent present in the lesions is highly acurate.[22] Other yeastlike organisms are distinctive in sections.

Under the microscope is seen an extreme amount of irregular acanthosis which results in irregular downgrowth of the epidermis. Throughout the epidermis are miliary abscesses with polymorphonuclear leukocytic cells, giant cells, erythrocytes and debris. The blastomycete may be either within the giant cells or free in the abscess. In the subepidermal layers the cellular reaction may be acute or chronic. Giant cells here are usually few, and the infiltrate is

made up of lymphocytes, epithelioid cells, plasma, and mast cells.

The pathological reaction often resembles tuberculosis and the finding of the organism is the only way to differentiate between blastomycosis and tuberculosis. The *B. dermatitidis* appears in tissue as a single-budding, spherical yeastlike cell with a thick refractile outer wall. It may be found in miliary abscesses (either within or without giant cells), between the epidermal cells, or scattered through the corium. There is a varying degree of spongiosus in the prickle-cell layer. Epithelial whorls and pseudo-epitheliomatous hyperplasia may be present. This feature may be misleading and if the appearance of the budding cells is not noted a diagnosis of superficial epithelioma may be erroneously made.

The organism may be grown in pure culture from the minute deep seated miliary abscesses at the margins of the verrucous lesion. In blood agar, or dextrose agar at 37°C, the growth is yeastlike, remains wrinkled, waxy, and reproduction occurs by the budding process similar to those seen in lesions. On Saboraud's glucose agar media at room temperature the growth is cottony, moldlike, and grows brown with age. The branching, filamentous hyphae produce small, ovoid, lateral spores which are either sessile or on short stalks.

A comparative study of cultures of *B. braziliensis* and *B. dermatitidis* has shown them to be of sufficiently similar characteristics to place them in the same genus.[23] Their differences culturally and clinically are considered of a specific rather than a generic importance.

As previously mentioned, intraperitoneal or intravenous inoculation may be used as an aid to diagnosis. Following inoculation into the peritoneal cavity infection develops in the liver, lungs, spleen, and lymph nodes in about three weeks. Fresh material from these lesions will reveal the budding organism.

THERAPY

The multitude of treatment regimes which are advocated speaks for the fact that no single medication or combination of methods is the one of choice. Clinical response has been claimed for ether in oil instilled rectally,[24] intensive sulfonamide therapy,[25] penicillin,[26] sulfapyridine solution locally,[27] and many others. On the other hand, penicillin and streptomycin have been reported to have no effect on *B. dermatitidis* and perhaps to even stimulate it.[28, 29]

Only the smallest of lesions may be surgically excised. Roentgen therapy alone has been advocated. It is claimed that x-ray stops activity in spreading borders, eliminates secondary infection, hastens regression of the chronic inflammatory reactions, and prior desensitization is not required. Large dosages of potassium iodide and superficial x-radiation in combination with good general medical treatment is probably the regime of choice.

Before therapy with potassium iodide is instituted it should be determined by complement fixation and skin testing whether the patient is hypersensitive to the organisms. Patients with positive skin tests should be desensitized before being treated with iodides. Smith[30] states that with neither positive skin tests nor complement fixation antibodies, the patient should be actively immunized with a heat-killed vaccine before iodide treatment.

Prognosis depends partly on extent of infection and partly on the immunologic response. It is best in patients with positive skin tests and no serum antibodies, and poorest in presence of a high titer complement fixation and a negative skin test.[30] Recurrences are common. However, the clinical response under iodides alone is so good that the addition of superficial radiation should completely erase the disease.

REPORT OF CASES

A most excellent and exhaustive report on blastomycosis was made, in 1939, by Martin and Smith.[31, 32] Their study included 80 proven cases from the literature and 13 new ones from North America. Since that report the first proved case of Gilchrist disease (cutaneous) in Europe has been claimed.[33] Seven other cases have been reported in Canada.[34-35] Other series have

also been recorded in the United States.

In a twenty-year period, 1930-1949, 58 proven cases of primary cutaneous blastomycosis have been seen at Charity Hospital of Louisiana at New Orleans. All are previously unpublished. Approximately 300 cases of deep mycoses of all types were reviewed. Of this number only 58 with diagnosis of blastomycosis were substantiated, presented cutaneous lesions which were cutaneous primarily, and in which the clinical description and history were adequate for presentation. In the 9 patients with systemic lesions also, the systemic spread was considered to have occurred secondarily.

Most of these patients had lesions on exposed areas of the body. In order of involvement were the head and neck, hands and forearms, and the legs to a much lesser extent. Our series shows significant numbers of patients in the occupational groups of day laborer and occupations related to wood or wood products (Table 1). Sixteen (or 27 per cent) gave histories of employment in lumber or related industries. Nine of these had received scratches or puncture wounds. Three others (not included in these 16) had received trauma from shrimptail, injury on plow, and scratch from garbage can lid.

The oldest patient in this series was 75 years and the youngest 9 years. Both had cutaneous involvement only. Two others were of school age. Martin and Smith have stated that no systemic infections have been described in a patient under 12 years or over the age of 70. However, the age distribution is considerably wider in infections essentially limited to the skin. Table 2 shows age incidence.

Males are much oftener infected with blastomycosis than are females. This fact is probably due to their more frequent exposure to infection by occupation, etc. In our series the ratio of male: female was 4.3:1 Martin and Smith reported a more extreme ratio of 9:1. Forty-eight per cent of our series was in the white race. A considerable portion of white patients were of foreign extraction with French and Italian predominating. Fifty-two per cent were Negro (Table 3).

Gilchrist disease is essentially limited to North America. It is reported in 28 states

TABLE 1
OCCUPATION

Domestic	8	Lumber Worker	9
Farmer	8	Other	6
Day Labor	22	Unknown	5
		Total	58

Patients with occupation related to lumber or wood products, or of injury caused by such—16

TABLE 2
AGE INCIDENCE

Age	0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	?	Total
Male			9	13	9	10	6			47
Female	1	2	3	1				2	2	11
Total	1	2	12	14	9	10	6	2	2	58

TABLE 3
RACE AND SEX

Race	Total	% Total	Male	% Male	Female	% Female
Total	58	100%	47	81%	11	19%
White	28	48%	22	79%	6	21%
Negro	30	52%	25	83%	5	17%

and Canada. As previously mentioned, a single case has been reported from Europe. Fifty-five of the patients in this series were residents of Louisiana; one each from Texas, Alabama and Mississippi. Analysis of the map of the State of Louisiana (Fig. 1) will reveal that all but 4 of the cases

GEOGRAPHIC LOCATION OF CASES OF BLASTOMYCOSIS
Indicates individual cases
Total cases shown on map
Rural Louisiana, but not located on map
Others (Texas, Alabama Mississippi)

Figure 1

which could be accurately located are from the southern parishes which border along the Gulf Coast and particularly along the lower Mississippi River. We feel that this possibly has significant meaning—this area is characteristically warm, low, humid, and abounds in timber, particularly cypress. Also a great portion is swamp or marshy land.

Table 4 shows how the diagnosis was substantiated in the cases in this series. Table 5 indicated that some patients sought medical care early in the course of the disease. However, a large number had lesions for longer than twelve months before being seen by a physician, and 9 patients did not seek medical aid until five years or more had elapsed after the appearance of the cutaneous lesion.

Some of the patients who had sought medical care were treated for furuncles, verrucae, epitheliomata, or syphilitic lesions. The three patients whom we have seen most recently had been under treatment elsewhere with diagnosis other than blastomycosis. These facts impress us that the physician in general is not alert to the presence of the disease.

Table 6 summarizes the types of treatment employed in these 58 patients. The number of patients in each grouping is not great enough to draw any definite over-all conclusions. However, it is noted that the patients who were treated by combined iodide and x-radiation regime responded well. The one death noted in this group was a generalized furuncle-type of infection and was caused by a fulminating toxemia.

SUMMARY

A brief review of literature and a summary of 58 previously unpublished cases of primary cutaneous blastomycosis has been presented. Nine patients in this series had cutaneous and systemic lesions. The systemic lesions were considered to be secondary to the cutaneous infection. Almost one-third of the patients were employed in lumber or related industries. The sex ratio is 4.3:1 more frequent in the male. It is suggested that the hot, humid climate, and the topography of this region may have a very

TABLE 4
DIAGNOSIS SUBSTANTIATED BY

	Cases		Cases
Direct exam. only	14	Autopsy only	3
Culture only	0	Biopsy and autopsy	3
Biopsy only	23	Direct, culture and autopsy	2
Direct and culture	2	Direct, biopsy and autopsy	1
Direct and biopsy	5	Direct, culture, biopsy and autopsy	1
Culture and biopsy	2	Unknown	2

TABLE 5
DURATION OF DISEASE PRIOR TO SEEKING MEDICAL CARE

Less than 1 mo.	1 mo.	2 mo.	3 mo.	4 mo.	5 mo.
4	2	8	6	2	....
6 mo.	**7 mo.**	**8 mo.**	**9 mo**	**10 mo.**	**11 mo.**
....	4	....	....	....	2
1 yr.	**2 yr.**	**3 yr.**	**4 yr.**	**5 yr.**	**5 yr. plus**
4	6	4	4	1	8

Unknown—3

TABLE 6
REVIEW OF TREATMENT

	Cured	Improved	No Change	Died	Died - Other Causes	No Information - Lost to Follow-up	Cutaneous Lesions Only	Cutaneous and Systemic Lesions	Total
Iodides	4	4	1	6	2	....	13	4	17
X-radiation	5	1	....	....	....	1	7	....	7
Surgery	1	....	....	1	....	1	2	1	3
Iodide-Xradiation	7	6	....	1	....	2	16	....	16
Iodides-Surgery	....	....	1	1	....	....	1	1	2
Xradiation-Surgery	2	....	....	....	....	....	2	....	2
Surgery-Desinsitization	....	....	2	....	....	....	2	....	2
Supportive Measures Only	....	....	....	3	....	1	1	3	4
Type of Treatment Unknown	1	3	....	....	....	1	5	....	5
Total	20	14	4	12	2	6	49	9	58

definite bearing on the incidence of the disease here. Definite conclusions cannot be made as to the value of any one mode of treatment, although it appears that the combined use of iodides and superficial x-radiation was most effective.

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DISCUSSION

Dr. Chas. Barrett Kennedy (New Orleans): Blastomycosis has been too much neglected by the medical profession as a whole since it was first recognized as a clinico-pathological entity by a dermatologist, Gilchrist, fifty-eight years ago. I enjoyed Dr. Black's fine word-picture of the disease. I have always wanted to know what the incidence of this disease was, in our own endemic area. Doctors from other areas have often sought that information from us. Dr. John Lamb, who was just recently a guest of our Louisiana Dermatological Society, said that he had never had an opportunity to see so many cases of deep mycotic infection at any one time at a skin meeting, and repeatedly urged us to collect, publish, and review them. I regret that this paper is not being presented before a more diversified group.

A brief look at the very recent issues of the cumulative index shows increasing interest in this disease by other specialties. A recent issue of the Journal of Urology reports a case whose presenting complaint was pyuria, with the fungus first found microscopically in the wet prostatic secretion. No cutaneous lesions were present at that time, although they appeared later.

I agree with the authors that iodides and x-ray are the methods of choice in treatment. I have had little experience with desensitization. At the New Orleans Veterans' Hospital, one cases of blastomycosis with lung involvement was unimproved on 800 drops of iodides daily. Prodigiosin was of no value in this case, but 250 cc. of convalescent hyperimmune serum was of most decided benefit.

I note in Dr. Black's summary that he feels that the systemic lesions were secondary to the cutaneous ones. I have personally observed some of the cases presented in his series, and have felt very definitely that the reverse was sometimes true.

CURRENT THERAPY OF SEVERAL COMMON SKIN DISEASES*

R. H. ROBINSON, M. D.

LAFAYETTE

The past decade and a half have seen revolutionary changes in the practice of the art and science of medicine. In the thirties, the advent of the sulfa drugs changed radically the prognostic outlook of many infectious diseases. In the forties, penicillin, streptomycin, and other antibiotics widened greatly this already benefited field. Now, also, with adrenal cortex[1] hormonal studies we see radical changes in outlooks of allergic and some of the less clearly understood diseases, such as rheumatic fever and lupus erythematosus.

Dermatology and syphilology have advanced with the other specialties along these same lines. The practice of our specialty has seen marked improvements thanks to the above and other revelations.

SYPHILIS

To most of us syphilology is a fast disappearing specialty. In the past three years, I have seen but one chancre and three secondary manifestations of syphilis. An occasional old "burned out" case with still positive serology is referred for opinion and evaluation. Penicillin has changed the method of treatment of this disease from a horrible nightmare of years to a matter of a few days. Different authorities recommend widely differing schedules of treatment but one method widely agreed as effective is a schedule of penicillin injections of 6,000,000 units.[2] Some authorities recommend more, others believe that bismuth and arsenicals[3] are indicated. It should be emphasized that this is for primary, secondary, or early latent syphilis. Fever therapy is still used at some institutions for central nervous system[4] lues. Aureomycin,[5] chloromycetin, and terramycin have been used experimentally with encouraging results. Aureomycin seems to have shown the best record thus far. One should keep this in mind for the occasional penicillin sensitive indi-

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 30, 1952.

vidual but the new hypo-allergic[6] penicillin compounds have done much towards decreasing the number of reactions seen.

Prophylactic injections of penicillin after exposure have been[7] used extensively and positive serologic responses many months later are surprisingly low. Skeptics, however, warn that this procedure may only mask the disease and that serious visceral complications may not show up until many years later.

Syphilis is a disease that is apparently as old as modern civilization[8] and we must not shrug it off as being conquered until many more years of study are completed.

FUNGUS INFECTIONS

In this more or less subtropical country fungus infections are extremely common We see the epidermophytosis of feet, hands, tinea of groins, body, and tinea capitis almost daily. In my experience one of the greatest mistakes commonly made is to overtreat these conditions, resulting in secondary contact dermatitis and secondary infection—especially on feet and hands. Secondary infection should subside under soothing soaks and compresses, antibiotic ointments such as bacitracin, and sulfa and antibiotics by mouth if necessary. Tineal infections plus secondary infection may result in severe "id" reactions which may be very hard to control. Again, I say caution is the rule. After infection is controlled there are innumerable preparations on the market for tinea. I favor several fatty acid preparations such as sopronol, timofax, desenex, and others, such as quinolor compound ointment, vioform, and spergon. Asterol has been reported as very effective in scalp tinea.[9] We treat scalp cases by plucking individually infected hairs while they fluoresce under the Wood's light. Tinea versicolor responds well to ultraviolet light, hypo scrubs, and asterol creme.

ACNE

This scourge of youth has been markedly benefited in recent years. While no means limited to, it is to a great degree a disease of adolescents. The etiology of acne remains a little obscure but it is definitely a metabolic disturbance. Androgenic substances and the steroid hormones[10] frequently produce an acneiform eruption. The halogens also are commonly contributory. Some investigators are enthusiastic about stilbesterol[11] orally, estrogens orally or parenterally or incorporated into ointments. My personal experience with these has been rather disappointing. The sulfa and antibiotics are of frequent value in severe cystic acne. Andrews[12] frequently gives sulfa for long periods of time, either alone or combined with terramycin. Sulphur is still quite popular and several drug houses have put flesh tinted sulphur and resorcin compound ointments on the market.* Several companies make them in varying shades and strengths making the messy, greasy, colored and colorless ointments less necessary and improving morale and cosmetic appearance of the patient while under treatment. I do not believe this should ever be underestimated. Intra-derm[13] sulphur is a preparation in a wetting agent for greater absorption of deep lesions. I feel it has its place but do not use it routinely. Of course, the older treatments with x-ray and ultraviolet light must not be forgotten but should be reserved for the discretion of the specialist.

Seborrheic dermatitis, I mention along with acne, as the two are often associated. I routinely treat the scalp in acne, whether I see anything or not. The advent of selenium sulfide (selsun suspension) I find to be a valuable one, but plodding along with the traditional tar, sulphur, ammoniated mercury, salicylic acid, and resorcin preparations still are valuable. Here again the advantages are mainly in cosmetic advances. Colorless and well odored preparations add to cosmetic appearance and morale. Certain dietary restrictions, such as chocolates, cola drinks, and iodized salt, are of value here as in acne. Seborrheic dermatitis of ears, eyebrows, ali-nasi and other common sites is benefited by compresses and antibiotic ointments. At this time I am a little enthusiastic about polymyxin. Drying and soothing lotions help relieve pruritis.

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*Smith-Kline-French, Ar-Ex, Almay, Texas Pharmacal Co.

HAND AND OTHER COMMON ECZEMAS

The common ever present problem of the housewives eczema of the hands is certainly worthy of mention here but the causative features would fill a book in itself. Contact factors are common.[14] Etiologic agents such as detergents, soap powders, feathers, sprays, paints, varnishes, oils, greases, and others are elicited with careful history. Avoidance of these is the first step in therapy.[15] Here, I would like to inject a note of caution—never have a patient wear rubber gloves next to the skin. It is frequently irritating. We use cotton lined or linen lined gloves for this purpose. The treatments are numerous, but one successful way for a chronic hand eczema is to use soothing compresses such as domeboro soaks, vioform creme, or some other soothing agent, an antibiotic, if necessary, and a antihistamine. X-ray or ultra-violet in very small doses is sometimes helpful, but must be watched, as these are drifters and may sometimes be found to be receiving treatments from several sources at the same time. I will discuss this further.

SCABIES AND OTHER PARASITIC INFECTIONS

The greasy, messy ointments of sulphur and similar preparations used for these annoying conditions have to a large measure given way to newer and nicer to handle preparations. Scabies can be handled with a 20 to 30 per cent emulsion of benzyl benzoate. I personally do not use it because of the large number of reactions to it. C.S.C. produces a preparation call kwell. This is 2 per cent hexachlorocyclohexane in a water soluble base that is most effective and two to three applications cure most patients. I have also found it most effective in all kinds of pediculi. "Geigy" produces a preparation called eurax that I have found of value but by no means 100 per cent effective. It is of considerable benefit because of the antipruritic action, which is good. Occasionally it is necessary to use sulphur. I use sulphur ppt., balsum of Peru, of each 5 per cent in a water soluble base, and apply to entire body twice daily for four days. Some prefer to use lotia alba during the day because cosmetically it is nicer.

Lupus erythematosus to the dermatologist usually means not one but two diseases for practical purposes. The acute and subacute disseminate lupus erythematosus are catastrophic diseases and are strictly a specialized problem. Here also, the use of ACTH and cortisone has greatly improved the prognosis of many in this grave condition. This benefit is dramatic but too frequently there is relapse when the drug is withdrawn. Maintenance dosage must be found and continued.[16]

Discoid lupus is an entirely different condition. Most of these patients are in good general condition and the purpose of their visit is for cosmetic reasons. The lesions are almost invariably present on the face, scalp, neck, or exposed areas. Many of them have previously been treated as "ringworm." Avoidance of solar radiation as much as possible is the first thing I tell my patients. Local treatment such as freezing with dry ice or ethyl chloride may be helpful in old intractible lesions. I still employ bismuth, gold salts, arsenicals, liver concentrate and vitamin B_{12}, by injection. Particularly, sun-sensitive individuals are given antihistamines and protective cremes. A-fil and skolex are examples. I do not advise x-ray and never ultraviolet. Recently a report on calcium pantothenate and vitamin E came out.,[17] I have not used panthenol yet but intend to when it is stocked. We give vitamin E in doses of 100 to 1000 milligrams daily.

Pityriasis rosea is one of the common skin diseases and one commonly mistreated. It is easy to mistake the herald lesion for tinea, but once the generalized eruption occurs the diagnosis should be simple. Always exclude secondary lues by a routine serology in these cases. Ordinary tub baths are eliminated and spotty cleansing allowed. The occasional intensely pruritic case can have starch and soda or tar baths. A soothing lotion such as Schamburg's oily shake, or Pusey's calamine liniment are all that are needed locally. Antihistamines and sedation will control pruritis. Ultra-violet[18] light baths about twice weekly may abort or greatly reduce the duration of the condition

that normally lasts about two months.

Herpes zoster has been reported as successfully cleared with antibiotics such as chloramphenicol, aureomycin,[19] and terramycin. Also reported successful are injections of protamide.* Neither method has greatly impressed me, inasmuch as I have seen herpes develop while a patient was taking the antibiotic and be as explosive as any case without. Relief of pain is still our major problem and irradiation of dorsal ganglia is occasionally necessary. Herpes involving eyelids and face should also have an ophthalmologist in consultation because of the possibility of corneal ulcers accompanying the disease.

Herpes simplex of the recurrent variety is sometimes benefited by irradiation of the areas usually affected. Moccasin venom injections or repeated vaccination with smallpox vaccine may be also employed with success.

Contact dermatitis is one of the most commonly seen conditions in all branches of medicine. In medical men, technicians, nurses, and housewives, it is definitely an occupational disease. It may be due to soaps, detergents, rubber gloves, novocaine, penicillin, streptomycin, and a host of other causes. Careful checking and patch testing will sometimes reveal the cause but it must be remembered that a sensitized skin may react to a given agent, whereas a normal area on the same person may not. Lotions, ointments, cosmetics, flowers, plants are also frequent causes. Treatment is simple if you can find the offending agent but sometimes that is a person's livelihood. Protective gloves and ointments may help. If it is found to be a plant agent, a personalized vaccine may be given to desensitize the patient.

Drug eruptions are common, especially from penicillin, sulfa, and streptomycin. Any drug can cause reaction in a given individual but not in others. Some may cause so called "fixed drug" eruptions in which ingestation of this drug may cause an eruption in exactly the same area each time it is taken. Antipyrine and phenolphthalein are common offenders. In any of these conditions ACTH and cortisone may speed recovery and lesson morbidity. They are used in the same way as with dermatitis venenata or contact dermatitis.

We now come to one of the most pitiful of them all—infantile and/or atopic allergic eczema. These babies, first, are usually otherwise fat and healthy appearing, but the fat does not have a normal feel to it. An allergic diathesis may be manifested. There is usually history of eczema, hayfever, or asthma in the family—sometimes all three. There is often a marked sensitivity to eggs, wheat products, citrus fruits, and cow's milk products. There is also pronounced reaction to weather changes. It is discouraging to parents, patient, and physician to have a case of this doing well and flare up badly with a weather change, but that is one thing we cannot regulate so we have to do our best. Wool, feathers, and animal dander should be strictly avoided.

In severe weeping states soothing baths and compresses and mild lotions are used. Antihistamines and parenteral or supplementary vitamins are given to replace those eliminated in dietary restrictions. I like oatmeal baths as a soothing agent. Soap should not be used. The anogenital regions are cleansed with mineral oil which may be used to remove accumulated crusts. Later the coal tar preparations and or vioform may be used with success. Goat's milk or soy bean milk may be substituted for cow's milk. We often give ultra-violet, but rarely x-ray. Chronic cases of long standing may be benefited by the purchase of a home ultra-violet unit.

Lichen planus, dermatitis herpetiformis, and all pemphigoid conditions are merely mentioned. Their treatment and diagnosis may be quite difficult and should be reserved for the specialist. Other less common conditions are excluded because of a deficiency in time to explain them.

SUMMARY AND COMMENT

Again we say in dermatology, as in other fields of medicine, antibiotics, and sulfa

*Sherman laboratories.

drugs have found their place. Other new advances are mentioned.

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The Journal of the Louisiana State Medical Society

Established 1844

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

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SUBSCRIPTION TERMS: $4.00 per year in advance, postage paid, for the United States; $4.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

CHEST PAIN ASSOCIATED WITH PULMONARY HYPERTENSION

Viar and Harrison* have presented a highly informative discussion on chest pain in association with pulmonary hypertension. This type of chest pain is not uncommon and is likely to lead to the erroneous diagnosis of coronary arterial disease. In articles by previous observers, this pain was discussed in relation to pulmonary arteriosclerosis, mitral stenosis, and pulmonary embolism. The authors quoted have extended these observations on chest pain and have found that conditions other than those mentioned may be associated with chest pain not originating in the heart. They give six illustrative case reports typical of the several groups considered, and observe that most of the patients with pain have had either (1) lesion of the mitral valve; (2) certain congenital anomalies of the heart; (3) primary diffuse disorders of the lungs, especially asthma and emphysema; (4) disorders of the pulmonary artery, and more particularly embolism. The common factor which seems to exist in all of these is evidence of the increased pressure in the pulmonary vascular circuit. From the discussion of case histories presented it is apparent that patients with pulmonary hypertension may have pain which is very similar to that produced by disease of the coronary arteries. The location, radiation, quality, and intensity of the two types of pain may be indistinguishable. The pain may be brought on by exertion and relieved by rest. Some patients may have prolonged bouts of pain, enduring for hours or days. In the six case reports there were three patients in whom electrocardiographic changes were prominent. In two of these, autopsy revealed no evidence of coronary disease. The authors note that in years past the error of mistaking coronary hypertensive pain for angina has been repeatedly made. They note that the differentiation, while difficult, and occasionally impossible, can usually be made if the syndrome of pulmonary hypertensive pain is kept in mind, and if sufficient care and observation be undertaken. The authors stress the importance of searching for a condition capable of causing pulmonary hypertension; that is, in the four groups of conditions mentioned above. They note that because of the low pulmonary arterial pressure in the tetralogy of Fallot, pain has not been noted in this condition. They also observe that not all patients with disorders reasonably

*Viar, W. N., and Harrison, T. R.: Chest pain in association with pulmonary hypertension; its similarity to the pain of coronary disease, Circulation 5:1, (Jan.) 1952.

associated with pulmonary hypertension have the complaint of pain and it is rarely encountered in patients with pulmonary hypertension secondary to left ventricular failure.

In addition to these considerations, there are certain features which may serve as guides in differentiating the pain associated with pulmonary hypertension from that accompanying coronary disease. Long standing cough is present in many of the patients with pulmonary hypertension. Cyanosis, either persistent or intermittent, is apt to be present during episodes of pulmonary hypertensive pain. The opposite is apt to be true in patients with coronary pain. The pain of pulmonary hypertension is apt to be associated with dyspnea, while that of angina typically is not. Increase in pain on inspiratory effort may be present. This pain should be in the center of the chest rather than in the axillary areas. Presumably, this increase of pain may be due to the increased flow of blood into the chest during inspiration and a consequent rise in pulmonary pressure.

There is considerable variability in the duration of pulmonary hypertensive pain. By way of differentiation, the authors note that in coronary disease the pain frequently falls into one of three categories as regards duration: (a) pain lasting a few minutes only, induced by exercise and relieved by rest (angina); (b) pain lasting thirty minutes or longer occurring in repeated attacks and usually during recumbency, and soon leading to a fatal issue or to the typical clinical picture of myocardial infarction (status anginosus); (c) pain lasting several hours to several days and then disappearing completely (myocardial infarction). All of these patterns of pain duration may occur in the pain of pulmonary hypertension, but in addition, the pain of the latter condition may be associated with a steady substernal feeling of distention enduring for hours or days. The patient with status asthmaticus may have pain as long as the asthma persists.

Any evidence of right ventricular hypertrophy is suggestive of pulmonary hypertensive pain. Such evidence may be clinical, electrocardiographic, or roentgenologic. The authors feel that the presence of diffuse forceful pulsations over the whole left precordial area presents more evidence of right ventricular enlargement than is usually obtained by the other means.

Electrocardiographic changes may be clarifying in efforts at differentiation. The absence of electrocardiographic changes argues in some degree against myocardial infarction, but of course, not against angina. In cardiographic changes of cor pulmonale, pulmonary hypertension is suggested.

Evidences of tissue destruction, such as fever, leukocytosis, and rapid sedimentation rate, are expected in myocardial infarction, but they may also occur in pulmonary infarction. Striking relief of pain as the result of oxygen inhalation argues somewhat for pulmonary hypertensive pain.

The effect of drugs may be helpful in distinguishing the two types of pain. Adrenaline may induce or aggravate the pain of coronary disease and tends to relieve pulmonary hypertensive pain when bronchial asthma is the cause. Intravenous administration of aminophyllin may relieve either pain. Relief following oral or rectal administration is more likely to occur with pulmonary hypertensive pain. Nitroglycerine has minimal effect on pulmonary hypertensive pain. Its use helps to differentiate pulmonary hypertensive pain from angina, but not from myocardial infarction. The authors state that they are unwilling to make a positive diagnosis of angina pectoris in the absence of evidence of striking benefit from nitroglycerine.

As a group, the prognosis in patients with pulmonary hypertensive pain appears to be better than that for patients with pain from angina and coronary artery disease. Sudden death is less likely in the pulmonary group.

Consideration was given to the neurological aspects of the pain. It was suggested that the pain is similar to that of coronary disease because the nerve connections of the

pulmonary arteries are the same as those of the heart. It is also suggested that the increase of pain on effort is the result of increase in pulmonary pressure during exercise in disease states. The investigators showed there is a well marked rise in pulmonary pressure in persons with emphysema and mitral stenosis during exercise. It is probable from clinical observations and corollation with other data that the pain is produced on distention of the pulmonary arterial system. In support of this, may be cited an experience of another observer studying athletes who run short distances. Toward the finish, they put forth the utmost effort and during this sprint they experience sudden extreme chest pain. If this is unheeded they are apt to drop.

These observations on pulmonary hypertensive pain are most timely. In this day of increasing heart disease and of increasing public apprehension in regard to chest pain, the physician's diagnostic ability is taxed. Accordingly, realization of the significance and importance of pulmonary hypertensive pain will be most helpful.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1953 ANNUAL MEETING

Under the direction of Dr. C. J. Brown, General Chairman of the Committees on Arrangements for the 1953 Annual Meeting, which will be held in New Orleans, May 7-9, plans are underway for the convention this year. Following is personnel of the various subcommittees and members are requested to contact these doctors concerning any phase of the meeting in which they are particularly interested or if they have suggestions to offer in regard to arrangements for the meeting.

Advisory—Drs. H. B. Alsobrook, Edgar Burns, Boni DeLaureal, A. V. Friedrichs, Val Fuchs, M. M. Green, Roy B. Harrison, E. L. Irwin, Edwin H. Lawson, Shirley Lyons, Leon J. Menville, Waldemar Metz, D. J. Murphy, J. Kelly Stone, H. Ashton Thomas, N. J. Tessitore, E. L. Zander.

Badges—Dr. J. T. Brierre, Chairman; Dr. Maurice Campagna, Vice-Chairman; Drs. Carroll Gelbke, Chris Bellone, Edmond Mickal, Louis Cabiran.

Banquet—Dr. W. Robyn Hardy, Chairman; Dr. Earl C. Smith, Vice-Chairman; Drs. Henry Butker, E. Garland Walls, Richard L. Buck, John J. Signorelli.

Decoration—Dr. Sam Houston, Chairman; Dr. Paul L. Getzoff, Vice-Chairman; Drs. Lawrence H. Strug, Homer J. Dupuy, Oscar Blitz, Morris Shushan.

Entertainment—Dr. Charles B. Odom, Chairman; Dr. Felix A. Planche, Vice-Chairman; Drs. J. O. Weilbaecher, Jr., Joseph S. D'Antoni, Edward Matthews.

Finances—Dr. George H. Hauser, Chairman; Dr. C. W. Mattingly, Vice-Chairman; Drs. Lucien A. Fortier, John J. Irwin, L. S. Charbonnet, Jr.

Golf—Dr. W. C. Rivenbark, Chairman; Dr. Jack Strange, Vice-Chairman; Drs. H. Ashton Thomas, Max M. Green, Joseph A. Vella, Roy de la Houssaye.

Hospitals—Dr. Wm. J. Rein, Chairman; Dr. E. L. Leckert, Vice-Chairman; Drs. Ernest Celli, Mayo L. Emory, W. P. Gardiner.

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Lanterns—Dr. W. H. Gillentine, Chairman; Dr. Howard R. Mahorner, Vice-Chairman; Drs. Ambrose Storck, Edward W. Nelson.

Luncheons—Dr. Wm. H. Roeling, Chairman; Dr. Eugene H. Countiss, Vice-Chairman; Drs. Lawrence O'Neil, John Oakley,

Frank Kinberger, Edward J. Joubert, Max M. Hattaway.

Publicity—Dr. Nicholas Chetta, Chairman; Dr. Jules Myron Davidson, Vice-Chairman; Drs. Ralph Hartwell, Eugene Vickery, Nicholas K. Edrington.

Registration—Dr. Joseph Ciolino, Chairman; Dr. J. C. Burns, Vice-Chairman; Drs. Robert Gillespie, J. M. Ciaravella, Joseph M. Brocato, Anthony Failla.

Scientific Exhibit — Dr. Edgar Hull, Chairman; Dr. James K. Howles, Vice-Chairman; Drs. Henry Ogden, Henry Duhe, Howard Karr, Jose L. Garcia-Oller, Louis A. Monte.

Signs—Dr. Abe Mickal, Chairman; Drs. Benjamin Morrison, Henry Larose, Paul B. Lastrapes, Leon Hart.

Technical Exhibit—Dr. Morell W. Miller, Chairman; Dr. Robert Sharp, Vice-Chairman; Drs. Lloyd J. Kuhn, Esmond Fatter, Eruno Mancuso, I. W. Kaplan.

Transportation—Dr. John Colclough, Chairman; Dr. Hugh T. Beacham, Vice-Chairman; Drs. Vincent Culotta, B. F. Danton, Jr., J. L. Fischman.

Woman's Auxiliary—Dr. Edwin R. Guidry, Chairman; Dr. Joel B. Gray, Vice-Chairman; Drs. Louis Leggio, Philip J. Carter, Hyder F. Brewster.

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VOLUNTARY PENSION PLANS

The following comes from the Committee on Legislation of the American Medical Association. In December, 1952 the House of Delegates at its Denver meeting reaffirmed its position advocating the enactment of legislation of this type. Our members should request the senators and congressmen from Louisiana to support these two bills. All out effort was made by our Congressional Committee to have the "Keogh-Reed" bills passed in the 82nd Congress. These are good bills and should merit our attention and support.

"During the first week of the 83rd Congress bills were again introduced to permit tax deferments on amounts used to purchase retirement annuities. These measures, H.R. 10 and H.R. 11, which were introduced by Mr. Jenkins of Ohio and Mr. Keogh of New York, are identical with the "Keogh-Reed" bills of the 82nd Congress. The bills are designed to encourage the establishment of voluntary pension plans by individuals and to provide more equitable tax treatment for self-employed persons. The bills in their present form now include all the amendments suggested by the American Medical Association at hearings on the original legislation last May.

"Through amendment of the Federal Internal Revenue Code, the bills would allow physicians and other self-employed individuals to deduct from their taxable income those amounts used each year to finance restricted retirement plans. Employed persons not covered by existing pension plans also would qualify for the tax deduction privilege.

"Income received later during the years of retirement, either from pension funds or insurance annuities, would then be taxable under the prevailing rates. In other words, they provide tax deferment but not tax avoidance.

"Physicians, dentists, lawyers, architects, farmers, store owners and the many others who comprise the nation's self-employed have long been neglected in federal tax legislation relating to pensions. The purpose of these bills is to eliminate the discrimination and inequities existing under present tax laws. By extending the tax deferment privilege to the country's ten million self-employed, and also to millions of employees who are not covered by pension plans, this new legislation will give them the incentive to save for old age during their best earning years.

"Under Section 165 (a) of the 1942 Revenue Act, millions of employees covered by more than 16,000 approved pension plans are already receiving the benefit of pension tax deferment. They pay no tax during their working years on the employer's contribution to their retirement fund, even though it actually is extra compensation to the employee. After he retires, however,

the employee must pay the tax on the part of the retirement benefit which was financed by the employer.

"To provide a similar tax arrangement for self-employed persons, the bills would allow annual deductions of 10 per cent of earned net income, or $7,500, whichever is smaller. Total deductions during the taxpayer's life-time could not exceed $150,000. The funds excluded from taxable income would have to be paid either to a trust fund established by an association for the benefit of its members or to an insurance company as premiums for a retirement annuity contract. In either case, no income payments or cash refunds could be made before the age of 65 except in cases of total disability or death.

"The bills include a provision enabling persons already between the ages of 55 and 75 to make larger annual deductions than the basic 10 per cent or $7,500. This is a practical equivalent to the past service credits allowed in many employee pension plans. The bills also provide for a carryover of unused deductions for a period up to five years. This is designed to give equitable treatment to persons with extreme fluctuations in income.

"This legislation will be of particular benefit to physicians who go through a long and costly period of training and whose peak earnings are bunched into a comparatively short period of years when they are subject to high income tax rates.

"This legislation, which has the approval of the American Medical Association and twenty other national organizations, can be enacted by the 83rd Congress if physicians and all other self-employed persons work actively together for its passage."

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IMPORTANT

Those of you who may be interested in the chiropractic problem in our state would do well to secure a copy of "Science vs Chiropractic" published by the Public Affairs Committee, Inc. (22 East 38th Street, New York 16, N.Y.).

This article deals with the subject in a masterful manner, clearly outlining the unsupported claims of the chiropractic cultists and most forcibly presenting the proven findings exemplified by modern science as evidenced by known and accepted experimentation and verified by members of the medical profession, throughout this and other countries, in actual practice.

The advantages and disadvantages of licensing chiropractors are discussed quite fully, but in conclusion it states that whether chiropractors "are licensed or practice against the law, uninformed people will continue to patronize them just as they do bookmakers or fortune tellers and for the same reason; they do not realize that the odds are all against them."

This little booklet deals with the following subjects: basic science laws, chiropractic education, how the cult of chiropractic got its name, the chiropractic advertising campaign, legal hazards of chiropractic and where chiropractic and science disagree.

The pamphlet only contains twenty-nine pages and can be read by many of your patients in the waiting room or can be handed them to be read at home by them and by their friends. It will be a wonderful way to educate the public and support our all out campaign against these cultists, who at every session of the legislature try to have enacted legislation legalizing their practice in our state.

The cost of this pamphlet is only twenty-five cents and certainly each and every doctor should be only too glad to purchase a few of them for distribution among his patients, friends and legislators, thereby helping to keep these cultists out of our state, and it goes without saying that we sorely need your support in these efforts.

Thank you for your interest and cooperation.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

LOUISIANA HEART ASSOCIATION WILL MAKE GRANTS FOR RESEARCH

The Louisiana Heart Asscociation announces that it will welcome requests for grants in aid for research in cardiovascular disease and in related fields. The deadline for receipt of requests is named as April 1, 1952.

Application blanks should be obtained from the office of Louisiana Heart Association and requests addressed to Dr. Russell Holman, Chairman, Research & Fellowship Committee, Louisiana Heart Association, Inc., Room 301 Balter Bldg., New Orleans, La.

A.M.A. PRESIDENT LISTS MEDICINE'S MAJOR OBJECTIVES FOR 1953

With the dawn of a new year, Dr. Louis H. Bauer, Hempstead, N. Y., president of the American Medical Association, outlined a constructive nine-point program for what he called "the preservation of our American system of medicine." His nine points, directed to all physicians and to all component societies of the A.M.A. are:

1. Work with rural communities to establish facilities for physicians, so that we shall have a better distribution of physicians.
2. See that good medical care for the indigent is available everywhere, just as it is in some states.
3. Extend public health coverage to areas lacking it.
4. Develop plans for the care of the chronic invalid.
5. Expand our voluntary insurance program, not only to cover more persons, but to cover those over age 65 and those suffering from illness of long duration.
6. Clean our own house, by disciplining those physicians who are tarnishing the reputation of the whole profession by their unethical acts of overcharging, accepting kick-backs, and making commercial arrangements with pharmacists.
7. See that the public is protected so that they can always obtain the services of physicians.
8. Revitalize our county societies and make them leaders in their communities in all health matters.
9. Inculcate the newly trained physicians in the tradition and ethics of medicine.

A.M.A. HOUSE OF DELEGATES ACTION ON FEDERAL MEDICAL SERVICES

After long debate, the AMA's House of Delegates adopted a resolution stating that the fundamental consideration of limiting the care of veterans in Veterans Administration hospitals to the two following categaries is sound: (1) to veterans with peacetime or wartime service whose disabilities or disease are service-incurred or aggravated and (2) to veterans with wartime service suffering from tuberculosis or psychiatric or neurological disorders of non-service-connected origin, who are unable to defray the necessary hospital expenses.

The resolution further comments that to discontinue the provision of medical care and hospitalization in VA hospitals for the remaining groups of veterans with non-service-connected disabilities cannot be accomplished without the cooperation of Congress, veterans organizations and the medical profession. The House of Delegates recommended that the AMA meet with representatives of veterans organizations, the American Hospital Association, American Dental Association, the Department of Defense, and the Veterans Administration to discuss problems and work out a satisfactory arrangement . . . rather than take action at the present time.

PATIENTS IN MENTAL HOSPITALS

The number of patients in State and county mental hospitals continued to increase, according to preliminary information on the annual census for 1950 compiled by the Public Health Service of the Federal Security Agency. The census is based on reports submitted by 201 State and 112 county mental hospitals to the National Institute of Mental Health.

A total of about 600,000 persons were under supervision of these hospitals in the United States at the end of 1950, the census report indicates, as compared with slightly over 580,000 in 1949.

SIXTH ANNUAL POSTGRADUATE COURSE IN DISEASES OF THE CHEST

The Sixth Annual Postgraduate Course in Diseases of the Chest sponsored by the American College of Chest Physicians, Pennsylvania Chapter and

the Laennec Society of Philadelphia, will be presented at the Bellevue-Stratford Hotel, Philadelphia, Pennsylvania, March 23-27, 1953.

This course will emphasize the recent developments in all aspects of the diagnosis and treatment of chest disease. The course is open to all physicians; however, the number of registrants will be limited.

The tuition fee is $50 and applications will be accepted in the order in which they are received. This course has been approved for credits by the American Academy of General Practice. Applications should be sent to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

OAK RIDGE COURSE IN AUTO-RADIOGRAPHY

An advanced course in autoradiography and three basic courses in radioisotope techniques are course offerings of interest to medical and biological personnel planned this spring and summer by the Special Training Division of the Oak Ridge Institute of Nuclear Studies.

The autoradiography course will be held from June 15-25, and basic courses of four weeks' duration will begin on June 8, July 6, and August 10.

The autoradiography course will be the second to be given at Oak Ridge, an earlier one having been given in the summer of 1951. It is intended for research level personnel using or planning to use this technique in their research.

Application forms and additional information may be obtained from the Special Training Division, Oak Ridge Institute of Nuclear Studies, P. O. Box 117, Oak Ridge, Tenn.

THE AMERICAN CONGRESS OF PHYSICAL MEDICINE AND REHABILITATION

The 31st annual scientific and clinical session of the American Congress of Physical Medicine and Rehabilitation will be held on August 31, September 1, 2, 3 and 4, 1953, inclusive, at the Palmer House, Chicago, Ill.

Scientific and clinical sessions will be given on the days of August 31 and September 1, 2 and 3. All sessions will be open to members of the medical profession in good standing with the American Medical Association.

In addition to the scientific sessions, annual instruction seminars will be held. These lectures will be open to physicians as well as to therapists, who are registered with the American Registry of Physical Therapists or the American Occupational Therapy Association.

Full information may be obtained by writing to the executive offices, American Congress of Physical Medicine and Rehabilitation, 30 North Michigan Avenue, Chicago 2, Illinois.

SOUTHWEST ALLERGY FORUM TO MEET

The Southwest Allergy Forum will meet at the Hotel Muehlebach, Kansas City, Missouri, June 14-15-16, 1953. The sessions are devoted primarily to papers on practical aspects of allergy. Those interested in participating in the program are invited to write the president, Dr. Orval Withers, Bryant Bldg., Kansas City 6, Missouri.

Members of the local committee are (besides Dr. Withers): Drs. Cecil Kohn, R. Dale Dickson, Frederic Speer, Stanley Goldman, Ralph Hale, Herbert Rinkel, and Vernon C. Wiksten.

Reservations should be made early at the Muehlebach; they may be cancelled if unforseen circumstances prevent attendance.

For information write: Frederic Speer, M. D., Sec. Treas., 2601 Parallel Ave., Kansas City, Kansas.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

EASING OF LOUISIANA NURSE SHORTAGE PREDICTED

Speaking before the quarterly meeting of the Second District Medical Society's Auxiliary, Mrs. T. E. Strain, president of the State Medical Auxiliary, predicted a lessening of the acute shortage of nurses in the near future. She pointed out that the state auxiliary is encouraging young women who have shown an interest in nursing as a career. Future Nurses Clubs are beginning to spring up in Louisiana high schools, Mrs. Strain said, and point to many new possible additions to the nursing field. The state president also outlined plans for observing Doctor's Day and for assisting in programs of the American Medical Association.

Following her talk, the Second District Auxiliary, which is composed of members from Jefferson, St. Charles, St. John the Baptist, and St. James Parishes, voted to contact school superintendents in their communities to arrange for members to speak before the junior and senior classes of the high schools.

ESSAY CONTEST IS OPENED

The 1953 National Essay Contest for high school students, sponsored by the Association of American Physicians and Surgeons was opened this month in Jefferson, St. Charles, and St. John the Baptist parishes. The auxiliary to the Second District Medical Society is conducting the competition, which offers the winner a $25 prize and the winner's school a $15 contribution to its library fund. The best essays will be considered for state and national prizes.

ORLEANS PARISH

An all-out nurse recruitment program, which begins in high school, is the Woman's Auxiliary to the Orleans Parish Medical Society's New Year's gift to the community, inspired by the critical shortage of "Women in White" the seriousness of which none knows better than the auxiliary. Future Nurses' Clubs will be launched in junior and senior high schools on a city-wide basis early this year. The foundation work has already been made by Mrs. Edwin Guidry, the auxiliary's 1953 president, and Mrs. J. Theodore Brierre (a former nurse) who is Nurse Recruitment chairman. They have made personal appearances with student nurses from the five local schools, and shown films and distributed literature about the "Women in White" in a few of the schools. Ten auxiliary leaders (former nurses), one for each senior and junior high school in the city have been named to launch the Future Nurses' Clubs. Student Nurses from Charity, Hotel Dieu, Touro, Mercy and Baptist hospitals will tell the Future Nurses about their training. Sponsors will work in close cooperation with school and nursing authorities on curriculum requirements and programs. For the first time the auxiliary is sponsoring a nursing scholarship in 1953 to put a worthy student through training.

LAFAYETTE PARISH AUXILIARY

The Lafayette Parish Medical Auxiliary has had interesting and well-attended meetings for the first few months of the 1952-53 term.

Beginning the fall activities, a benefit style show was sponsored by the auxiliary. All funds received were given to the Parish Polio Fund. This amounted to $542.50. In October the Auxiliary entertained the state auxiliary president, Mrs. T. E. Strain, with an informal tea given in the home of Mrs. J. Boring Montgomery. Also in October "treat day" was held for the patients of the tuberculosis hospital on the eve of Halloween. At the November meeting, Dr. Thomas Arceneaur, dean of the college of agriculture of Southwestern Louisiana Institute, presented colored pictures of French West Africa which he himself had taken when he went there as agricultural consultant for the Mutual Security Agency. The pictures with their explanation were most interesting and educational. The December meeting was a delightful Christmas party, at which time members brought gifts to be given to the St. Vincent De Paul Society for distribution to children for Christmas.

BOOK REVIEWS

The Pathogenesis of Tuberculosis; by Arnold R. Rich, M. D. 2d ed. Springfield, Ill., Charles C. Thomas, 1951. pp. 1028, illus. Price $15.00.

In this second edition Dr. Rich has brought up to date all the known factors entering into the natural course of tuberculous infection. In doing this, there is presented bibliographic material from which more detailed knowledge can be obtained on any one factor. The chapters of the tubercle bacillus have been brought up to date in presenting the material on the relation of chemical constitutents to pathogenesis, the different types of tubercle bacilli and the significance of the variations in types of tubercle bacilli.

There are six chapters on the significance of virulence and native resistance with a survey of all the known factors which may influence resistance to tubercle bacterial infection. Following are three chapters on orientation on the principles of hypersensitivity as applied to tuberculosis and there is an excellent group of chapters on the mechanism of acquired resistance. The discussion on factors that influence resistance is quite inclusive and includes the evaluation of pertinent literature up to the time of publication. The discussion of factors responsible for the characteristics of tuberculous lesions brings one up to date with current concepts.

The significance of the material presented in the monograph is well understood in reading Dr. Rich's interpretation of its presence in the health of our civilization. The application of material presented can be of great importance to the practicing physician, as well as those in the fields of public health and preventive medicine, since the monograph presents concepts and evaluations which previously had not been integrated.

JOSEPH E. SCHENTHAL, M. D.

PUBLICATIONS RECEIVED

Rutgers University Press, New Brunswick, N. J.: The Literature on Streptomycin, by Selman A. Waksman.

W. B. Saunders Company, Phila.: Operating Room Technique, from St. Mary's Hospital, Rochester, Minnesota (4th Edit.).

Charles C. Thomas, Publisher, Springfield, Ill.: Introduction to Physiological Optics, by Armin Von Tschermak-Seysenegg and translated by Paul Boeder, Ph.D.; Practical Blood Grouping Methods by Robert L. Wall, M. D.; Vitamins for Blood Formation, by Thomas H. Jukes, Ph.D.; The Biochemistry of Gastric Acid Secretion, by Edward J. Conway, M. D.; The Pharmacology of Anesthetic Drugs, by John Adriani, M. D. (3rd Edit.); Body Temperature, by W. A. Selle, Ph.D.; Visual Anatomy: Thorax and Abdomen, by Sidney M. Friedman, M. D.; Analgesia and Anesthesia in Obstetrics, by J. P. Greenhill, M. D.; A Vitamin Digest, by Guy W. Clark; Acute Renal Failure, by John T. Maclean, M. D.; Gynecology, Diseases and Minor Surgery, by Robert J. Lowrie, M. A., M. D.